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ANNALS
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VOL. 46 MARCH, 1937 No. 1

I

THE SURGERY OF OTOSCLEROSIS*

PROFESSOR GUNNAR HOLMGREN

STOCKHOLM, SWEDEN

For the diminution of hearing in otosclerosis there is no generally accepted remedy, chiefly because of the fact that hitherto none of the various procedures have produced demonstrable results. Surgical methods may be considered an exception to this rule in as much as every operator who has sought to solve the problem has obtained immediate and noticeable improvement in hearing as soon as a fistula was produced leading to the labyrinth, in different locations.

This occurred for the first time as Kessel, in 1876, removed the stapes in order to replace the footplate with a (movable) scar membrane. Passow obtained the same result in 1897 by trephining the promontory. In these cases improvement in hearing was not permanent but lasted only a few days.

The opposition to these experiments was strong and quite universal. At the Congress in Rome, in 1894, leading otologists (Politzer, Moure and Cozzolino) condemned stapes extraction as useless and dangerous. At the Congress in Paris, in 1900, Siebenmann and Botey took exception to the method as ineffectual, dangerous to hearing and to life, and essentially futile in view of the nature of the disease. An equally adverse stand was taken by Denker, in 1904, in his monograph, "Otosclerosis."

*Read before the III International Congress of Oto-Rhino-Laryngology at Berlin, August, 1936.

A decade passed before the question was again taken up, this time by Bárány. Supported by his well-known theory of the function of artificial ear drums, Bárány suggested a fistula into the posterior vertical canal through which better hearing might be established and aseptic conditions maintained. Both aims were accomplished, but the results persisted only a few days.

In 1913 Jenkins opened the horizontal semicircular canal in two cases. He covered the fistula in one case with a Thiersch graft, in the other with a flap from the external meatus, and obtained in both cases an undoubted improvement in hearing. One patient became completely deaf soon thereafter and the other so nearly so that Jenkins abandoned the method.

With a view to bringing about a more lasting result by covering the fistula at operation with an elastic membrane, thus producing primary healing, Holmgren in 1917 suggested making the fistula at the upper part of the anterior vertical canal, removing the bone between the dura and the fistula and thus bringing about primary healing between the two. In one case operated upon by this method, the immediate hearing result was good but of short duration. A proposal by the same author to produce a still thinner scar through excision of the dura over the fistula with primary union with the arachnoid was not attempted.

Somewhat later, Bárány suggested a new technic in two stages. In the first the processus mastoideus was opened with gouges and the hollow was filled with fat. Some weeks later, at a second sitting, the horizontal semicircular canal was trephined and a small morsel of fat brought in apposition to the fistula to keep it open. The effect on the hearing was satisfactory but lasted only two weeks.

In 1920, Holmgren suggested a new technic to the Oto-Rhino-Laryngological Congress in Paris. This operation was carried out under magnifying instruments (Gullstrand's loop and Zeiss' binocular microscope). The fistula, originally established at the promontory, was covered with mucoperiosteum previously turned to one side. This method gave good but only temporary results.

Two years later, Holmgren attempted a fistula into the horizontal semicircular canal, which he covered immediately with the mucoperiosteum of the ampulla. This time the hearing was much improved and it was only after a few weeks that it again disappeared. In several cases, however, the improvement, though not great, was lasting. By this method, which could point to some results, however

small, otologic patients with increased bone conduction were operated upon with the result that they were able to resume occupations which they had been forced to abandon; in all, some thirty-five in a period of fifteen years. It was observed by some of these patients that hearing in the unoperated ear became progressively worse but in the ear operated upon remained stationary. The worse of the two ears was therefore operated upon, which is now in many cases the better one. Sourdille observed and published such a case.

I shall not dwell upon Wittmaack's and Heyninx's well-known operations, as these do not anticipate an improvement in hearing but only an arrest of symptoms.

Since 1924, Sourdille attacked the problem with a unique intelligence and technical facility. I shall not enter into a discussion of his technic nor his results, as he is appearing personally before this Congress. His experience now embraces numbers of cases which, albeit after repeated tedious and technically difficult operations, have attained undeniable and enduring improvement. Stimulated by the optimism engendered by Sourdille's results, I have recently reopened the problem.

Since my patients formerly operated upon obtained a good—in fact, of late a very good—primary hearing, although the end results were minute, I assume that this was due to the fact that not merely a fibrous but a bony covering of the fistula had occurred. I attempted to cover the fistula according to Thiersch, a method formerly suggested by Floderus, later by Jenkins, possibly by others. One of these patients has had for many years an undoubted improvement which is not great but sufficient to permit him to resume his former occupation which before the operation he had had to abandon. This method had the disadvantage that the patient retained behind his ear a permanent opening lined with squamous epithelium, which now and again required attention. Therefore, I abandoned the method.

The method which I have finally evolved has now given considerably better results over a period of one and one-half years. Before I describe this more closely I wish to present several cases.

REPORT OF CASES

CASE 1.—E. K., 50-year-old teacher, Kiruna. Examined in 1928, 1935 and 1936. One brother and two cousins are deaf. There has been a progressive diminution of hearing on both sides, for the past thirty years on the left, seventeen on the right, with paracusis Willisii and intermittent tinnitus. The drums are irregularly thickened, somewhat retracted after otitis at the age of 17. Fixed. X-ray shows normal pneumatization except at the apex.

6. 5, 1936.—Hearing test.

Right		Left
0	Whisper	ad concham
0.2 meter	Conversation	1 metre
C128	Lower limit	C128
5 seconds	C128 air conduction	15 sec. (normally 120)
45 seconds	C128 bone conduction	45—50 sec. (normally 45)

6. 6, 1936.—Operation for otosclerosis on right ear, in which the reduction of hearing had reached the highest degree and the progression was going on most rapidly. Healing per primam. Length of stay in hospital, eleven days.

7. 14, 1936.—Hearing test. Right ear.

Whisper	0.15 meter
Conversation	10 meters
Lower limit	C64
C128 air conduction	33 seconds (normally 120)
C128 bone conduction	75 seconds (normally 45)

The patient was operated on because of the progressive reduction of hearing, which threatened to keep her away from her work.

Case 2.—A. D., age 33, woman farm laborer, Fränsta, Central Sweden. Examined April-June, 1936.

Heredity uncertain. Progressive reduction of hearing for six years in the right, for two years in the left ear. Treated without effect, lately with thyroxin.

Nervous patient operated on for Basedow's disease.

Normal position and normal mobility of the tympanic membranes. X-ray, normal pneumatization, no pneumatization of apex.

5. 14, 1936.—Hearing test.

Right		Left
0	Whisper	0.3 meter
0.5 meter	Conversation	2.5—3 meters
C128	Lower limit of tone	C64
3 seconds	C128 air conduction	20 sec. (normally 120)
45 seconds	C128 bone conduction	45—50 sec. (normally 45)

5. 15, 1936.—Operation for otosclerosis in the right ear, in which the reduction of hearing had reached the highest degree. Healing per primam. Length of stay in hospital, 28 days.

7. 14, 1936.—Hearing test. Right ear.

0.15 meter	Whisper
10 meters	Conversation
C64	Lower limit of tone
40 seconds (normally 120)	C128 air conduction
65 seconds (normally 45)	C128 bone conduction

The comparatively long stay in hospital was in this case due to the lability of the nervous system of the patient.

CASE 3.—K. K., age 50, foreman, stone cutter, Stockholm. No hereditary otosclerosis. Bilateral progressive reduction of hearing for thirteen years. Singing in his ears like the sound of a waterfall, feeling of intense heaviness in his head. Tympanic membranes somewhat thickened. X-ray: Normal pneumatization, no pneumatization of the apex.

10. 10-22, 1935.—Hearing test.

Right		Left
0	Whisper	0
0.20 meter	Conversation	0.50—1 meter
C 256	Lower limit of tone	C 128
0	C 128 air conduction	
65 seconds	C 128 bone conduction	65 sec. (normally 45)

10. 22, 1935.—Operation for otosclerosis. The operation was carried out on the right ear, in which the reduction of hearing was most pronounced.

6. 8, 1936.—Hearing test. Right ear.

0.20 meter	Whisper
4 meters	Conversation
C 256	Lower limit of tone
0	C 128 air conduction
15 seconds	C 128 bone conduction

July-August, 1936. Conversation, 4 meters.

Apart from the improvement in his hearing the patient's general state of health has improved considerably. His former feeling of intense heaviness in the head has quite gone off. The singing in his ears has not entirely disappeared but is greatly reduced.

CASE 4.—D. S., age 41, ship owner, Oslo. Examined June and September, 1935.

No inherited otosclerosis. Bilateral progressive reduction of hearing for ten years. Various treatments by several physicians without effect. Bilateral exostosis. Tympanic membranes normal.

Handles of the malleus movable. X-ray: Normal pneumatization, no pneumatization of the apex.

9. 6, 1935.—Hearing test.

Right		Left
9	Whisper	
0.20 metre	Conversation	0.40 meter
C 256	Lower limit of tone	C 128
0	C 128 air conduction	1—2 sec. (normally 120)
40 sec.	C 128 bone conduction	35 sec. (normally 35)

9. 7, 1935.—Operation for otosclerosis. The operation was carried out on the right ear, which presented prolonged bone conduction as well as the greater reduction of hearing and the more rapid progression. Healing per primam. Length of stay in hospital, nine days.

3. 30, 1936.—Hearing test. Right ear.

Whisper	0.40 meter
Conversation	10 meters
Lower limit of tone	C64
C128 air conduction	10 seconds
C128 bone conduction	45 seconds

Before the operation the patient could hardly hear, even with a bone conduction telephone held to the left ear. Since the operation this apparatus has no longer been needed. The patient hears now well enough to carry on his large business. He takes part in meetings and social life. He feels no giddiness whatsoever in daily life. He is a keen sportsman and skis without difficulty.

CASE 5.—A. K., age 22, woman. Examined August 1935 and January-August, 1936. No inherited otosclerosis. Bilateral progressive reduction of hearing during the past year. Painful singing in her ears. Paracusis Willisii. Treated without effect. Tympanic membranes normal, normally movable. X-ray; Normal pneumatization, no pneumatization of apex.

5. 5, 1936. Hearing test.

Right		Left
0	Whisper	0
0.5 meter	Conversation	0.3 meter

5. 6, 1936.—Operation for otosclerosis on the left ear.

7. 14, 1936.—Hearing test. Left ear.

Whisper, 0.2 m.
Conversation, 10 m.

Since 7. 14, a rapid reduction of hearing in the ear operated upon.

8. 3.—Hearing test.

Right		Left
0.1 meter	Whisper	0
0.5—1 meter	Conversation	0.2—0.5 meter
C128	Lower limit of tone	C128
7 seconds	C128 air conduction	4 sec. (normally 120)
40 seconds	C128 bone conduction	40—45 sec. (normally 45)

8. 3.—Operation for otosclerosis (revision) on the left ear. Healing per primam. Length of stay in hospital, fourteen days.

8. 13.—Hearing test. Left ear.

Whisper	0.2—0.3 meter
Conversation	>10m. meters
Lower limit of tone	C32
C128 air conduction	40 seconds (normally 120)
C128 bone conduction	70 seconds (normally 45)

CASE 6.—S. K., age 48, merchant, Oslo. Bilateral progressive reduction of hearing for sixteen years. Continual singing in the ears. Paracusis Willisii. Treated without effect. Used an ear trumpet. Tympanic membranes normal. Handles of the malleus immovable. X-ray, normal pneumatization. No pneumatization of apex.

6. 17, 1936.—Hearing test.

Right		Left
0	Whisper	0
0.6 meter	Conversation	0.6 meter
C128	Lower limit of tone	C128
9 seconds	C128 air conduction	13 sec. (normally 120)
80 seconds	C128 bone conduction	70 sec. (normally 45)

6. 17, 1936.—Operation for otosclerosis on the right ear. Healing per primam. Length of stay in hospital, nineteen days.

6. 26, 1936.—Hearing test. Right ear.

0.40 metre	Whisper
27 meters	Conversation
C32	Lower limit of tone
65 seconds	Air conduction
80 seconds	Bone conduction

The indications are simple. With a view to results, all cases may be operated upon who have a pronounced reduction in hearing with a raised lower tone limit, negative Rinne, prolonged or at least not shortened Schwabach, a normal drum membrane and roentgenologically normal middle ear, i. e., cases of high grade otosclerosis without labyrinthine degeneration.

A pronounced reduction I regard as one which renders the patient unable to follow his vocation; generally speaking, one meter or less conversation. One can be certain of improving this limit. As my records show, prolonged bone conduction is not necessary for good prognosis nor is an entirely normal drum membrane.

The technic is equally simple, although still in the stage of development. In principle, it is a decompression of peri- as well as endolymph by the production of a fistula which is covered with a soft, resilient tissue.

At one operation, which I performed in 1920, I made the following observation. I operated under the microscope on the promontory, made a flap of mucoperiosteum, with its base forward, and pushed this flap forward with a small raspatory. This brought forth a hairline stream of clear labyrinthine fluid from a pre-formed fine lumen definitely anterior and somewhat inferior to the fenestra ovalis. This lumen was so small that it could not be seen even with the magnification of ten diameters. There was an immediate and pronounced improvement in the patient's hearing without the labyrinth having been uncovered at any point. This preformed canal may have been the fistula ante fenestram described by Otto Mayer and Gordon Wilson.

Through this operation it was determined that a decompression of the labyrinthine fluid can improve the hearing. At every operation for otosclerosis that I have done under the microscope since then I have observed that the fluid escaped as soon as the fistula was begun and before the endosteum, so far as could be determined through the microscope, was perforated. Sourdille made the same observation. This decompression of the perilymph I believe can play a role in the sudden improvement which the fistula brings about.

The same holds good for decompression of the endolymph. If one, before reaching the semicircular canals, uncovers the anterior lateral wall of the saccus endolymphaticus there is in some cases—not in all—a prompt and perceptible, if not a high grade improvement in the hearing.

The operative technic was as follows: Under a local anesthetic a wide antrotomy is performed with most careful hemostasis. Bleeding from the bone is slight, nevertheless it is carefully stopped with bone wax. The anterior lateral wall of the saccus endolymphaticus is now uncovered, when the hearing often increases to about two meters for conversation. Now, preferably with a chisel, fistulas are made to the horizontal and posterior vertical canals, sometimes also to the anterior vertical. Each fistula is made fairly long, four to five millimeters, the endosteum is removed without injuring the membranous labyrinth and the fistulas are widened as much as possible. As soon as the fistula reaches a canal, the hearing increases promptly and constantly; in fact, all patients afterward hear conversation at the greatest distances that my operating room, together with the adjoining room, permits—that is, about ten meters. Now the aditus ad antrum is enlarged and a soft prothesis is laid into the antrum which on the one hand prevents the collection of blood and wound secretions and on the other supplies an elastic wall for the fistula and which should crowd the fistula somewhat to prevent it from closing during the period of healing. As protheses I have employed various substances: a thin rubber bladder (filled with air, with physiologic salt solution or with fat), a piece of gutta percha or, more recently, fat. In order to prevent the undesired growth of fat to the membranous labyrinth I have in several cases coated the antrum wall with leaf gold before the fat prothesis was laid in. The incision was closed with Michel clips. The operation is done under aseptic precautions and healing per primam follows.

Although it will not be possible here to recount the details of the various operations, I shall touch upon a few of the more important ones.

Since February, 1935, I have operated upon thirty-four patients with various modifications of this technic; of these, thirteen cases (ten women and three men) received fat protheses with leaf gold over the fistulas. Among these cases there occurred no severe complication and no death. Only in the beginning was there some vertigo. In individual cases this was quite pronounced for about a week. Usually it is mild, even in the beginning. The vertigo disappeared at latest after one month.

Those patients who previous to the operation complained of tinnitus state that this disappeared entirely or almost entirely with the establishment of the fistula. In those cases in which the auditory improvement is reduced and eventually disappears during healing, tinnitus recurs. There appears to be, in these cases, a certain parallelism between reduction in hearing and tinnitus.

The improvement in hearing at the time of operation is constant and astonishing. In that moment in which the fistula is established, the hearing increases for conversation in practically all cases to at least ten meters, rarely somewhat less. When the patient, after resting a few hours following operation, is examined in one of the corridors of the hospital, he repeats conversation heard at a distance of twenty, thirty and sometimes even forty meters. During the period of healing, the hearing is reduced in some cases to the preoperative state; in two cases it was reduced even further. In the majority of cases, in spite of this, a greater or lesser improvement in hearing is attained which in certain cases is even progressive. It is only after many years' observation that one may speak of a permanent improvement in hearing; until then I will content myself with stating that in the thirteen patients treated with fat prothesis and gold leaf, eleven have improved, eight of them to a pronounced degree. A reliable estimate of hearing requires experience as well as care and exactitude. It is well known that hearing in otosclerosis undergoes wide fluctuations. The general status of the patient, passing nervousness, variations in the weather and so on, affect it. The surroundings, the examiner and the method of conducting the test naturally play an equally important role in the result.

My cases have all been examined by two examiners, by the same methods, once in a soundproof room and once under ordinary surroundings. Aside from this, audiograms were made in the camera silenta. In no case was the hearing recorded without many confirmatory tests having been made.

The operative technic here given is simple. It recommends itself as safe and not very time-consuming and yields in practically all cases a definite improvement in hearing which, during healing, is again somewhat reduced. Over a period of one and one-half years a certain improvement in hearing has persisted in the majority of cases, in most of them quite pronounced. Years must elapse before this may be demonstrated to be permanent.

This work is the result of long experience with experimental animals. How shall the fistulæ be made in order to produce the greatest primary improvement in hearing and how shall they later be handled in order to achieve the greatest permanent good? Those are the two important questions demanding attention. Once these are answered, one may expect that the surgical management of otosclerosis will be successful in the hands of those who can maintain the necessary asepsis.

II

INTRACRANIAL COMPLICATIONS OF OTOGENOUS THROMBOSIS OF THE LATERAL SINUS*

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AND

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Thrombosis of the lateral sinus as a consequence of otitis media and mastoiditis is a lesion whose clinical and pathologic characteristics have come to be well known to otologists. The nature of its intracranial complications together with the mechanism of symptom production, on the other hand, are still poorly understood by pathologists, otologists and neurologists alike. This is probably due to the fact that opportunity for study of any large series of cases is unusual. Furthermore, in autopsied cases the pathologic picture presented is often bizarre and complicated, therefore difficult of interpretation. Finally, insofar as the meninges and brain are concerned, the interpretation of symptoms of the consequent intracranial lesions demands attention by those conversant with the anatomy, physiology and pathology of intracranial structures.

In this study it is our purpose briefly to survey the disturbances in structure and function responsible for neurologic symptoms and the various symptom-complexes resulting therefrom. A short review of the essential anatomy and physiology will serve as an introduction to the study proper.

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†It has been the writers' opportunity and privilege to have access to clinics and laboratories of the Los Angeles County Hospital in their efforts to study the various intracranial complications of otitis media and mastoiditis. We are deeply obligated to our colleagues in otology, Doctors Detling, Homme and their associates, for the courtesy shown us in inviting us to study their cases of otitis media presenting symptoms of intracranial extension. We are also obliged to Dr. Newton Evans, pathologist-in-chief, his assistant pathologists and the residents in pathology for permitting us to make a more critical study of the meninges and brain in the fatal cases of otogenous thrombosis than the press of their own duties would permit.

Essential Anatomy.—The lateral sinuses, lying between the two layers of the dura, are large venous channels which drain most of the blood from the intracranial space. For purposes of description, each sinus is divided into two parts. The posterior horizontal portion grooves the inner surface of the occipital bone while the descending or sigmoid portion, together with the jugular bulb, lies in intimate contact with the mastoid cells throughout its extent, separated from these cells only by a thin plate of bone, the sinus plate.

The lateral sinus receives the superior and inferior petrosal sinuses, which in turn drain the cavernous sinus and the afferent veins which drain the anterior portion of the cerebellum on one hand and the petrous bone on the other. Deserving particular attention is the fact that the sigmoid portion of the sinus receives the venous outflow, either directly or indirectly, from the upper portion of the middle ear, the antrum and the adjacent mastoid cells and labyrinth. By way of the inferior anastomotic vein (vein of Labbé), connection is made with the superficial middle cerebral vein and its afferents. Because of this rather direct connection between the genu of the lateral sinus and the "central" system (the superficial and deep middle cerebral veins which usually drain into the cavernous sinus) as well as the more roundabout way of the superior longitudinal sinus, the entire dorsolateral surface of the cerebral hemisphere is exposed to infection, once this has gained entrance to the lateral sinus. If the right side is involved, infection may locate in the upper part of either hemisphere since, in the average case, the right lateral sinus drains the upper part of both hemispheres, as this channel is a direct continuation of the superior longitudinal sinus. The left lateral sinus, on the other hand, is continuous with the straight sinus and therefore drains the interior of the brain. (For illustrations showing anatomic distribution of the dural sinuses and their afferent veins see Courville and Nielsen (1935, c-Fig. 6; 1937, Fig. 3).)

The horizontal portion of the sinus drains the adjacent portions of the cerebellar hemispheres and the occipital lobes.

The cerebral and cerebellar veins which empty into these more rigid dural sinuses are thin walled, without muscular coat and without valves. To reach the sinuses, they must traverse the subarachnoid and the subdural spaces, exposing these spaces to infection when the sinus becomes thrombosed. It is these anatomic characteristics of the lateral sinus and its communicating channels that account to some degree for the peculiar pathologic manifestations observed following infection secondary to otitis media and mastoiditis.

Essential Physiology.—The lateral sinus carries the greatest portion of venous blood from the intracranial space through the jugular bulb into the internal jugular vein. Since these channels and their afferent veins are without valves, the venous outflow is quite direct. This flow, however, is under negative rather than positive pressure in the system. The venous blood is pumped out of the skull by the action of the heart rather than evacuated by the force of gravity alone. The flow is also slowed by the numerous bends, twists and angulations in the course of the venous channels. It is also interfered with by trabeculations which run across the lumens of the larger sinuses. In the case of the superior longitudinal sinus, the blood has to ascend when the body is in the erect position and also against the inflow from the superior cerebral veins.

The lack of actual valvular obstruction makes it possible for the blood current to be entirely reversed within the system under certain circumstances. As a result of coughing, straining or vomiting, the current may be temporarily reversed, an alteration which is favored particularly by the presence of the inferior anastomotic veins which permit the backing up of blood from the region of the genu directly to the dorsolateral surface of the cerebral hemispheres. Therefore any foreign particle which, in the meantime, has gained entrance into the system, may be carried backward over this communicating system and may be deposited anywhere over the dorsolateral surface of the brain. In case of complete obstruction by a thrombus, passive reversal of the current may likewise be responsible for retrograde extension of infection to relatively distant parts of the brain.

It is a well known principle of inflammation that in acute conditions the possibility of blood-borne infections is markedly increased since the local venous channels are widely dilated. In chronic processes, on the other hand, because of organization of the inflammatory process with occlusion of vessels, direct invasion of vascular channels is rather unlikely. It is now well known that following an acute otitis media, especially in infancy, there may be a direct invasion of the veins draining the middle ear with extension into the intracranial space (acute primary otogenous meningitis) or extension into the general circulation (primary otogenous septicemia). The possible venous pathways by which infection may directly or indirectly reach the lateral sinus are as follows: (a) direct extension may be by way of the veins draining the middle ear, antrum or mastoid cells or from the lower part of the tympanum, (b) indirect extension may be (1) by way of the veins draining the tympanum, the antrum or the labyrinth or adjacent labyrinthine cells into the superior or, less likely,

into the inferior petrosal sinus. Indirect extension may also occur (2) by way of the mastoid emissary vein from a subperiosteal abscess. (3) Infection extending anteriorly into the pericarotid venous plexus may ultimately reach the jugular bulb into which these veins empty.

A general septicemia or bacteremia may result from an invasion of the small veins, either primarily, such as occurs predominantly in infancy and early childhood, or secondarily after gross thrombosis of the sinus. We have come to believe that a primary invasion may occur with acute otitis media, when under suitable circumstances a retrograde extension may result in lodgment of bacteria in the brain without the formation of an intermediary thrombus. To be sure, when there is a gross thrombosis of the lateral sinus and its afferent channels extending to the point of the cerebral lesion, the pathway of extension is quite obvious.

The lesions produced by thrombosis of the lateral sinus are extremely variable and depend upon (1) the pre-existing anatomic situation, (2) the virulence of the infecting organism, (3) the suddenness with which the channel is occluded, (4) the extent of thrombosis and (5) the character of local venous lesions.

The existing anatomic situation has long been recognized as an important factor in intracranial complications. In infancy and childhood the small venous channels draining the middle ear are larger than in adult life and the circulation is more active. Therefore, extension into the brain by way of these channels is much more likely at this age and frequently results in vascular lesions of the brain. Such lesions vary considerably in their character and may be of minor degree, such as local edema or red softening (nonsuppurative encephalitis) on one hand to actual inflammatory lesions, as suppurative encephalitis, subdural abscess, meningitis or actual abscess formation on the other. The development of this sort of a situation would predicate either the existence of an inferior anastomotic vein which empties into or very near the genu of the lateral sinus or a retrograde extension by way of the superior longitudinal sinus.

As to the virulence of the infecting organism, it is quite obvious that the more virulent the infection, the more likely it would be for infection to invade promptly the regional vessels and result in a meningitis. A less virulent infection, on the other hand, would probably result in a more chronic lesion, possibly an abscess or gross thrombosis of the lateral sinus itself. If the endotoxin of the infecting organism has marked histolytic properties, subdural or subarachnoid hemorrhage may result (Courville and Nielsen, 1934).

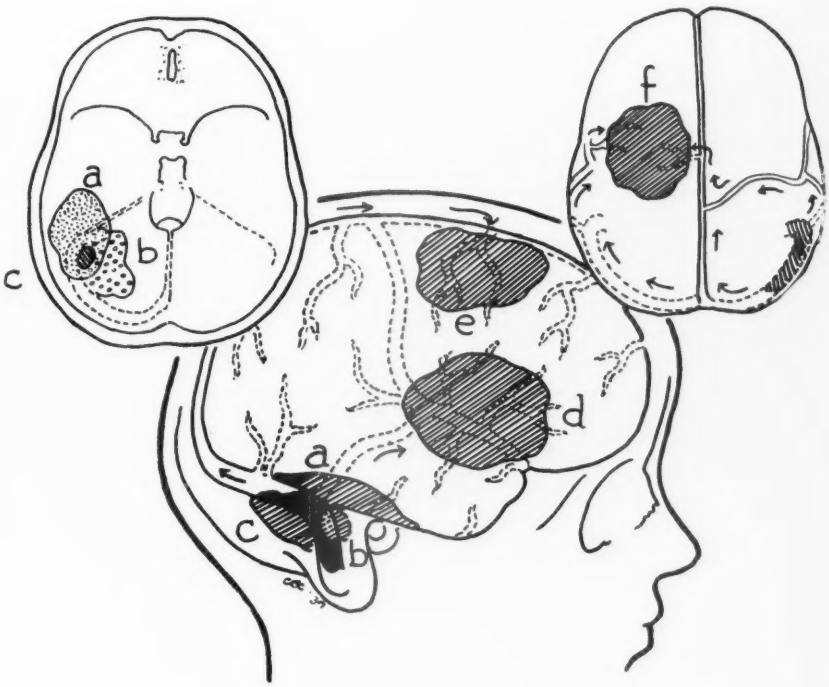

Fig. 1. Diagramatic drawing showing the possible locations of otogenous subdural abscess. They may be the result of direct extension (adjacent subdural abscess) above (a) or below the tentorium (b and c). This group of subdural abscesses may or may not be secondary to thrombosis of the lateral sinus. Distant subdural abscesses are the result of retrograde extension by way of communicating venous channels. These may be located on the same side (d and e) or the opposite side (f) as the thrombosed lateral sinus. The arrows point the possible venous routes traveled by the infection. (From "Pathology of the Nervous System," Pacific Press, 1937.)

It is evident from the study of certain cases that the suddenness with which the channel is occluded has something to do with the production of cerebral symptoms. For example, if a jugular vein is ligated for a presumed thrombosis of the sinus when such is not the case, certain transitory symptoms and signs of a cerebral lesion not infrequently result, probably the effect of a disordered circulation. In a great majority of cases, on the other hand, when an inflammatory thrombus slowly develops with a progressive occlusion of the channel, no cerebral symptoms result. As a matter of fact, it is now known that complete occlusion of the lateral sinus may occur without neurologic manifestations of any sort, the collapsed vessel being found at autopsy when the patient dies years later of some other cause.

The extent of the thrombus at times has something to do with the type of the lesion produced. In most cases of local thrombosis the development of any cerebral lesion is not dependent upon the size of the thrombus and, in most cases, no cerebral lesion occurs whatever. In some instances of local thrombosis, to be sure, small focal areas of red softening have been noted in the occipital lobe or more commonly in the adjacent portion of the cerebellum. On the other hand, with extensive retrograde thrombosis which occurs particularly in infancy and early childhood the thrombus extends into the adjacent veins, into the superior longitudinal sinus or into the straight sinus and at times into the opposite lateral sinus. Extensive external or internal red softening of the brain may result therefrom (Ehlers and Courville, 1936). In other cases, abscess formation in the parietal or frontal lobes may result from such a complex involvement of the venous system.

The character of the local venous lesion is of importance from the standpoint of the changes resulting in the meninges and brain. In many cases it is very likely that the lesion originally present as a result of retrograde embolism or extension clears up by the time of autopsy or operation. Little or no effect of such a lesion is evident from the standpoint of external examination. Many of these temporary lesions are undoubtedly of the character of a localized edema which clears up in the course of a few days or weeks without leaving any permanent residuals, either anatomically or clinically. Only when the alteration is sufficient to cause a local softening (non-suppurative encephalitis) or is complicated by infection, so as to produce an actual suppuration does the anatomic lesion become more or less persistent and produce pronounced cerebral symptoms. If the thrombus in the lateral sinus becomes infected, meningitis may result if the infection extends into the adjacent subarachnoid space, or, if

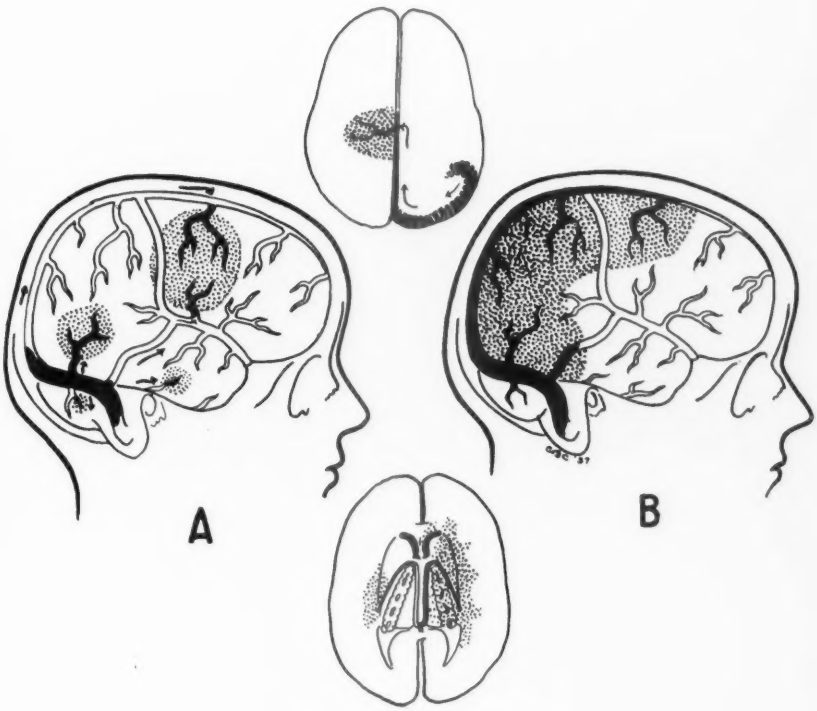

Fig. 2. Diagramatic drawing showing possible locations and extent of areas of red softening (non-suppurative encephalitis). A, focal areas of softening. Arrows show route of extension to softened region. B, extensive cerebral softening due to retrograde extension into the inferior and superior cerebral veins which occurs in infancy. Upper insert shows softening in contralateral parietal lobe. Lower insert shows softening in basal ganglia following retrograde extension into internal cerebral veins via the straight sinus. (From "Pathology of the Nervous System," Pacific Press, 1937.)

there is only a perivenous reaction, there may be local reactive changes in the meninges with pleocytosis but without definite meningeal infection. On the other hand, if venous occlusion results in red softening, the local infarction may become infected to form an abscess. These abscesses may be solitary or multiple and be located in almost any part of the brain.

The anatomic character of the abscess produced is such as to differentiate it from ordinary metastatic abscesses resulting from arterial extension, as from the lung or heart. These abscesses, following retrograde extension by way of superficial veins, are superficial in location, that is, they lie just beneath the pia mater and a layer of altered and attenuated cerebral cortex to which it is more or less intimately attached by its capsule. The abscess is usually fairly large, circumscribed and as a rule not well encapsulated. It is to be distinguished from typical metastatic abscesses of the brain which usually lie deep in the centrum, well beneath the cortex.

The incidence of intracranial complications secondary to thrombosis of the lateral sinus varies considerably. The various possibilities are noted in the accompanying table (Table I) which includes our findings in a study of 15,000 consecutive autopsies. It is impossible, of course, to be absolutely sure of the exact incidence of all lesions produced by invasion of the lateral sinus. Those stated include only those in which the sinus was found to be grossly infected and do not include those in which the extension occurred by retrograde embolism by way of these channels but without gross affection of the channel.

We may find, therefore, as a result of thrombosis of the lateral sinus, a wide variety of intracranial lesions, some transitory and benign, others of serious and often of lethal character. Among the first group may be included focal venous infarctions with meningeal and cortical edema, inconsequential foci of red softening and serous or reactive meningitis. Among the latter are to be included subdural abscess and hemorrhage, septic meningitis, gross areas of red softening and cerebellar or cerebral abscesses (Courville and Nielsen, 1934).

In this series of 15,000 autopsies, there were forty-three cases (0.3 percent) of otogenous thrombosis of the lateral sinus. In thirteen cases no other intracranial lesion was found. In fifteen cases a septic meningitis was also present. In ten cases other venous channels were also thrombosed. An abscess of the brain was found in seven cases (cerebellum 5 cases, temporal lobe 1 case and parietal lobe 1 case). Extradural (perisinus abscess) was found in two cases and subdural abscess in two cases. Subdural hemorrhage was found in four cases.

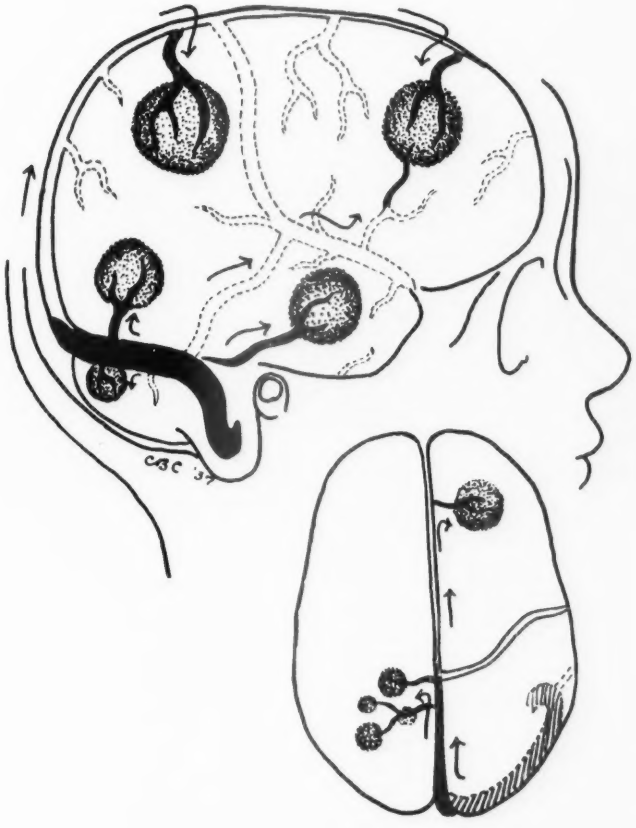

Fig. 3. Diagramatic drawing showing possible locations of "distant" otogenous abscesses of the brain. Arrows show routes of infection in each case. The abscess may be located in either the same or opposite side to the source of infection. The lateral sinus is thrombosed in a rather high percentage of cases. (From "Pathology of the Nervous System," Pacific Press, 1937.)

TABLE I

OTOGENOUS THROMBOSIS OF THE LATERAL SINUS AND ASSOCIATED INTRACRANIAL LESIONS IN A SERIES OF 15,000 AUTOPSIES

No.	Autopsy No.	Age	Sex	Causative Lesion	Mastoid Affected	Sinus Affected	Associated Intracranial Lesions
1	82	46	M	A or C?OM	L	L	O
2	188	?	M	A or C?OM	L	L	O
3	755	44	M	APOM	R & L	R & L	Thrombosis both cavernous, superior inferior petrosal, superior and inferior longitudinal sinuses. Subdural abscess. Right temporal lobe abscess. Meningitis.
4	1351	40	M	A or C?OM	?	?	O
5	4133	1	M	APOM	R & L	L	O
6	4295	6	F	APOM	R & L	L	O
7	4374	37	M	APOM	R	R	Thrombosis of the cavernous sinus. Right parietal abscess.
8	4516	7	F	APOM	R & L	R	O
9	4583	15	M	CPOM	L	L	O
10	4601	2 m.	F	APOM	R & L	R	Subdural hemorrhage.
11	5308	4 m.	F	APOM	R & L	R	Subpial hemorrhage.
12	5390	9	M	APOM	R	R	O
13	5961	15	M	APOM	R & L	L	O
14	6253	13	F	APOM	L	L	Extradural abscess.
15	6507	9	M	APOM	R & L	R	(Thrombosis of jugular bulb.)
16	7139	6 m.	M	APOM	R & L	R & L	Thrombosis superior longitudinal sinus.
17	7150	4	F	APOM	R & L	R & L	Thrombosis superior longitudinal sinus.
18	7164	1	M	APOM	R & L	R & L	Thrombosis superior longitudinal sinus.
19	7433	11	M	CPOM	R & L	R & L	Thrombosis of the cavernous sinus. Septic meningitis.
20	7518	25 d.	M	APOM	R	R	Extradural abscess. Thrombosis of superior longitudinal sinus.
21	8361	5	M	APOM	R	R	Thrombosis of cerebellar veins with softening and hemorrhage. Septic meningitis.

22	8685	58	F	CPOM	R & L	R & L	O
23	8742	17	M	APOM	L	L	Septic meningitis.
24	9228	15	M	CPOM	L	L	Septic meningitis. Cerebellar abscess.
25	9558	31	M	APOM	L	L	Septic meningitis. Cerebellar abscess.
26	9614	3	F	APOM	R	R	Subdural hemorrhage.
27	9641	9	F	CPOM	L	L	Thrombosis of inferior petrosal sinus.
28	9767	15	M	ↄ OM	L	L	Septic meningitis.
29	9844	33	F	CPOM	L	L	Septic meningitis. Cerebellar abscess.
30	10014	19	M	CPOM	R	R	Septic meningitis. Subdural hemorrhage.
31	10017	9	F	APOM	L	L	Septic meningitis.
32	10361	57	M	APOM	R & L	L	Septic meningitis. Subdural hemorrhage.
33	10528	8	M	ↄ OM	R & L	R	(Extension into jugular veins.)
34	11067	28	M	CPOM & M	R	R	Subdural abscess Left cerebellar abscess.
35	11139	23	F	CPOM & P Mastoid	R	R	Septic meningitis. Osteomyelitis of right petrous bone.
36	11705	2	F	APOM	R & L	L	Necrosis of dura.
37	12113	1	F	APOM	R & L	L	Thrombosis of the straight sinus and both veins of Galen with red softening of both thalami.
38	12632	8	M	CPOM & APM	L	L	Septic meningitis. Left cerebellar abscess.
39	12893	5	F	APOM	R & L	R	Pachymeningitis externa and interna.
40	13372	1	M	APOM	R & L	R & L	O
41	14082	7	M	APOM	L	L	O
42	14315	5	M	APOM	R	R	Septic meningitis.
43	15051	48	F	APOM & M	R	R	Septic meningitis.

SYMPTOMATOLOGY

In view of the theme outlined above, that spread of infection may readily occur through the medium of the superficial cerebral veins, it is clear that otogenous thrombosis of the lateral sinus can give rise to any intracranial complications. For the sake of organization the subject is presented as follows:

1. Recoverable damage due to stasis.
   - A. Symptoms when thrombosis of one lateral sinus is the essential lesion.
   - B. Symptoms when thrombosis of the superior sagittal sinus is added or is an isolated lesion.
2. Non-suppurative encephalitis.
3. Meningitis.
4. Encephalic abscess.*
   - A. Symptoms indicating temporal location.
   - B. Symptoms indicating cerebellar location.
   - C. Symptoms indicating parietal location.
   - D. Symptoms indicating frontal location.
   - E. Symptoms indicating occipital location.
5. Subdural abscess.
6. Spinal fluid findings.

1. RECOVERABLE DAMAGE DUE TO STASIS

The occurrence of transient cerebral symptoms due to stasis is more dependent upon pathologic physiology than on pathologic anatomy, although at the time of the symptoms microscopic examination might show anatomic changes. Certain mechanical factors play an important rôle in their production. These are the relative sizes of the venous channels affected, the efficiency of collateral circulation and the suddenness of the occlusion.

With regard to the relative sizes of the venous channels affected, it is well known that there is marked variability in the lateral sinuses. If a small one is suddenly occluded, there may be few if any symptoms, whereas if the larger one had been affected instead, paralysis might have resulted.† With reference to collateral circulation, the

*The lobar distribution of distant abscesses is largely dependent on the relative volumes of the lobes. The authors have dissected a brain into lobes, excluding the basal ganglia and the brain stem. The relative weights were as follows: Frontal 36, parietal 23, temporal 22, cerebellar 12, occipital 7 per cent.

†It is this variability which mitigates the value of the Tobey-Ayer test for thrombosis of the lateral sinus. Pressure applied to a small jugular vein may fail to cause a rise in the manometric fluid. In this way the test has led to erroneous interpretation of thrombosis.

presence and course of the anastomotic veins of Trolard and Labbé make considerable difference to the superficial circulation when a sinus is suddenly occluded. It is true that in a few reported cases the collateral circulation has been sufficient that even bilateral jugular ligation has been survived.

The suddenness of the occlusion is also of great moment. One author has gathered four cases in which death resulted from ligation of one jugular vein in operation for carcinoma. This is a very unusual event and the fatalities were ascribed to the suddenness of the occlusion and the fact that it was the larger vein which was ligated.†

The occurrence of transient cerebral symptoms from thrombosis of the lateral sinus has been recognized for many years. In his monograph of 1877, Nothnagel stated that depression of the mental faculties, increasing to sopor and coma, could occur. He considered, however, that, "disturbances of other kinds which often occur, such as paresis and paralysis, convulsions, hyperalgesias and the like" were due to meningitis and lesions of the brain. At that time it was generally agreed that there was usually meningitis accompanying all cases of sinus thrombosis. When thrombosis of the superior sagittal sinus occurred, it was thought to be marantic and not otogenous.

As early as 1888, Gowers stated that cerebral symptoms due to thrombosis of the lateral sinus were not definite, while convulsions shifting from the first to the second side were characteristic of occlusion of the superior sagittal sinus. He outlined in some detail the symptomatology of each of the sinuses. However, with the newer knowledge of intracranial complications of otitis media and the mechanism of spread of infection, one must consider the idea of a separate syndrome for each sinus entirely too naive and the problem must be studied anew. To show this clearly we shall present first cases from the literature and from our own experience in which thrombosis of the lateral sinus was the essential or only lesion.

A. Symptoms When Thrombosis of One Lateral Sinus Is the Essential Lesion.—Change in so fundamental an element as the personality itself has been reported by Mann (1930) and so profound a higher psychic disturbance as a frank psychosis by Rejtö (1932). Stupor is a somewhat common symptom and must occur at some time during each case when death eventually results. Beck (1931)

†We have had the opportunity to study a case in which the left jugular vein was ligated and the corresponding sinus packed when thrombosis had not occurred. A marked aphasia resulted and remained until the circulation was re-established four days later.

specifically mentioned coma. The simultaneous toxemia may account for general mental symptoms and we hesitate to ascribe such symptoms to involvment of a certain portion of the brain.

Aphasia is not rare but most men reporting cases of aphasic symptoms from thrombosis of the lateral sinuses have not taken the trouble to work out in detail the specific type of aphasia observed. Some have contented themselves simply with the word "aphasia". However, Herlinger (1932) specifically diagnosed amnesic aphasia, Mann (1930) motor aphasia, and Neubauer (1924) sensory and motor aphasia in their cases. In the last named instance, the thrombosis was on the right side, from which aphasia should not result. The vascular disturbance probably extended around or across to the other hemisphere.

The question of convulsions is closely associated with that of hemiplegia, the former being only the irritative stage of the latter. Unqualified convulsions are not infrequently reported and occasionally also jacksonian attacks. Beck reported the latter type in 1931 (affecting, however, the ipsilateral side) associated with hemiplegia of the side opposite the thrombosis. Birsner and Wolff (1930) reported twitching of the contralateral face. Neubauer (1924) has reported hemiplegia of the ipsilateral side (the lesion being contralateral). These observations are valuable in showing that when the symptomatology clearly depends upon thrombosis of a sinus, one must not invariably expect to find a focal lesion of a surgical nature even though symptoms pointing to such a lesion seem definite. As a rule, however, when hemiplegia occurs, it affects the limbs opposite the thrombosis as one would expect.

Frank cerebellar symptoms have been observed by Weise (1930) and Hofmann (1930) and hypotonia and diminished reflexes by Podestá (1932). Beck (1931) observed the symptom of falling backward, but this does not of necessity indicate a cerebellar lesion.

Signs referable to the cranial nerves are numerous. Choked disks have been observed by Birsner and Wolff (1930), Schenke (1927), Weise (1930), Neubauer (1924), Beck (1931), Podestá (1932) and many others. Babbitt (1925) saw it in mild degree. Uhthoff [cited from Alexander (1929)] found eyeground changes in 52 per cent of cases. In Schenke's case blindness of one eye resulted and the vision was impaired in the other eye. Nystagmus both toward [Herlinger (1932)] and away from the thrombosis (Weise, Neubauer, and Hofmann) has been observed. In the last two cases it was rotary. Pain in the distribution of the trigeminal nerve was reported by Mann in 1930, and palsy of the sixth has often been observed. From throm-

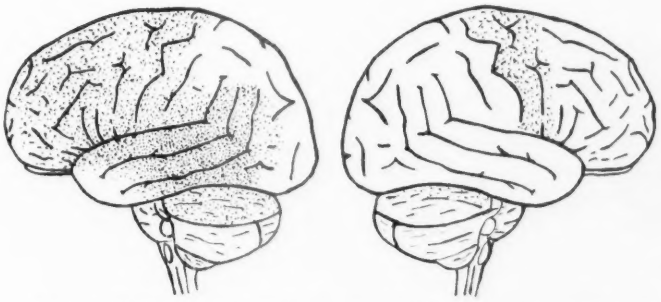

Fig. 4. Distribution of cerebral areas involved in nine cases of otogenous thrombosis of the lateral sinus. The distribution was judged by clinical symptoms. All lesions are illustrated as arising from the left ear. Note that there was considerable spread to the opposite side.

bosis of the jugular bulb Kümmel has reported involvement of the ninth, tenth and eleventh. Paralysis of the extra-ocular muscles is not uncommon (cases reported by Kümmel).

Ohnacker (1932) reported seeing four cases presenting the picture of a cerebral abscess.

If one were to outline on the surface of a brain the areas involved from occlusion of the lateral sinus alone, one would find that hardly a portion of the cortex remains unaffected. This applies not only to the ipsilateral, but to the contralateral side as well. In figure 4 we have indicated by the stippled areas the sections of cortex which were apparently responsible for the symptoms noted in nine cases of occlusion of one sinus from the postmortem records of the Los Angeles County Hospital. The wide distribution is striking.

Our own observations in these nine cases verify much of that which has been recorded above and we are able to add a few points. M. W. P. (Case No. 271-979), a white girl of six years, showed no focal cerebral symptoms but was delirious, and B. S. (Case No. 40-107), a white boy of nine years, showed only irrationality, but a Mexican boy of thirteen (Case No. 90-678) presented carphology in addition. In a case previously reported by the authors (1934), a boy of seventeen years, complained of a severe left-sided headache, pain in the left eye, and was irrational from thrombosis of the left lateral sinus. A right homonymous hemianopsia then developed with tendency to conjugate deviation of the head and eyes to the left (paralytic phenomenon), inequality of the pupils, amnesic aphasia

and elevation of the disks. The aphasia became practically complete, and as the patient became stuporous, hemiplegia developed. At autopsy only thrombosis of the left lateral sinus was found.

In the case of D. L. D. (Case No. 198-480), a boy of five years, with only a thrombosis of the right lateral sinus, there were first ipsilateral then contralateral convulsions of the jacksonian type. (There was, to be sure, a small subdural cerebellar hemorrhage, but this gave no symptoms.) In another very striking case in a man of 58 years in which autopsy showed only thrombosis of the left lateral sinus and sigmoid extending into the jugular vein there were the following symptoms: Pain in the left side of the face, vomiting, tortuous veins in the fundi, ipsilateral headache, spastic limbs and contralateral Babinski sign plus repeated attacks of coma and sensory aphasia. In all of the cases cited above the brain tissue showed only reversible changes, proved in some cases by recovery.

B. Symptoms When Thrombosis of the Superior Sagittal Sinus Is Added or Is an Isolated Lesion.—Thus far we have considered only the symptomatology when the essential lesion is thrombosis of the lateral sinus. There are cases in the literature in which in addition to the lateral there was also thrombosis of the superior sagittal sinus. The second case of Schenke is one in point. This patient, a man of 45 years, developed hemiplegia which disappeared entirely the next day. The following day convulsions appeared and the paralysis returned. Postmortem examination showed no meningitis but thrombosis of lateral, sigmoid and superior sagittal sinuses. In Rinaldi's patient who was observed from the beginning through every phase from thrombosis of the lateral to the occlusion of the superior sagittal sinus, convulsions never occurred and paralysis was limited to one side of the face. In Leidler's case, also verified, jacksonian seizures occurred during the stage of recovery. The recovery was, however, not prevented by the fact that the thrombus in the sagittal sinus could not be removed. In Doyle's second case, the patient had convulsions both jacksonian and general with stupor and aphasia.

In 1931, Chalier and Naussac reported a case of isolated thrombosis of the superior sagittal sinus, and, after studying the three cases described by Lanois, they came to the conclusion that there were two types of syndromes resulting from occlusion of the superior sagittal sinus, a fulminating form with general cerebral symptoms and a progressive form with ascending hemiplegia.

The material available to us from the Los Angeles County Hospital gives us four cases with autopsy in which the lateral sinus, one or both, and the superior sagittal were thrombosed. One of these

patients, a boy of one year, showed only meningeal signs. He died of pneumonia. There was never any paralysis or even a jacksonian seizure. Another child, only six months old, had generalized convulsions, decerebrate rigidity and a bilateral paralysis of the abducens. The other two showed the so-called superior sagittal syndrome, i. e., alternating jacksonian seizures, the one ending in hemiplegia.

In two other cases, with isolated thrombosis of the superior sagittal sinus one showed the characteristic alternating jacksonian seizures, but in the other no convulsions were ever recorded and only strabismus and meningeal signs were present. In the last case, a child of ten months presented at autopsy an area of softening in the right posterior parieto-occipital region. It is thus clear that one cannot depend upon convulsions to diagnosticate occlusion of the superior sagittal sinus.

A critical comparison of the cerebral symptoms which one encounters in thrombosis of the lateral sinus with those enumerated above as occurring when the superior sagittal is involved alone or in association with the lateral, presents the striking fact that the symptoms are indistinguishable. The one symptom or syndrome which has been held as distinguishing the lateral from the sagittal involvement, absence of alternating convulsions in one and their presence in the other, may occur in either one; and thrombosis of the one or both may occur without convulsions. This leads to the inescapable conclusion, that it is not the thrombosis of the sinuses which causes the symptoms. It is, of course, the effect of venous stasis on the cerebral cortex.

Isolated thrombosis of the petrosal sinuses is so rare that little consideration need be given to it. Such a condition usually leads to involvement of the cavernous sinuses and obstruction of this dominates the picture clinically. We have nothing new to add to the extensive work of Alexander on this subject. He has shown that the cavernous sinuses may be occluded without the condition being evident clinically and that in certain cases there are only a few signs, and these are not referable to intracranial complications. So far as these are concerned one may assume that paralysis of the extra-ocular muscles, meningeal signs and choking of the disks are the only neurological manifestations of this condition.

As a result of a study of the literature and the autopsy records on our own cases, we have come to the conclusion that the cerebral symptoms seen in occlusion of the various sinuses are not the direct result of thrombosis of the sinuses but are secondary to stasis of the superficial cerebral veins. The surgeon cannot, of course, treat the

cerebral veins. He must decide whether the lateral or perhaps the superior sagittal sinus is involved, and by removing the source of infection facilitate recovery.

It is well known that a thrombosed but uninfected lateral sinus does very little if any harm. Leidler's case shows that the superior sagittal sinus may also be thrombosed if not infected and still result in no serious cerebral damage.

2. NONSUPPURATIVE ENCEPHALITIS

Beside these transient disturbances of cortical function due to alterations in the circulation, thrombosis of the lateral sinus may also result in softening without suppuration in various situations. Probably the first to report this condition but without a discussion of its pathogenesis was Oppenheim (1897), who reported extension of otogenous encephalitis to the ipsilateral frontal lobe. Since that time a great many authors have studied the condition, chief among whom are Borries (1932), F. Voss (1902), O. Voss (1928) and Grinker and Stone (1928). The present authors reported a case with residuals under the heading of "Pseudo-abscess" (1934) and have discussed the subject in another study concerned with the cerebral manifestations following acute otitis media in infancy and in early childhood (1935). The report of Bodechtel and Richter (1933) deals with the same condition. It is noteworthy that in these conditions as in the cases without actual encephalitis there may also be extension to the frontal lobe, the parietal lobe and even to the other hemisphere. Extension to the parietal lobe is especially common in infancy and young children. Non-purulent encephalitis may cause paralytic manifestations and leave residuals of permanent type just as purulent lesions may. In fact, Borries dismisses the symptomatology of non-purulent encephalitis with the statement that the symptoms are those of abscess of the brain. We have found this to be essentially true, even bradycardia being present during the early stage due to increase in the intracranial pressure. Spontaneous recovery alone differentiates the non-purulent from the purulent encephalitis.

3. MENINGITIS

In the interests of economy of space a description of meningitis will be omitted since its symptomatology is already well known.

4. ENCEPHALIC ABSCESS

In so brief a presentation as this must be, it is impossible to give the subject of abscess of the brain the attention which it deserves. Only the briefest summary can be presented. Some general points of interest should be recorded. In the first place when abscess of the

brain develops in the presence of thrombosis of the sinuses, it is extremely difficult if not impossible for anyone absolutely to determine the fact. It is possible for cerebral abscess to develop alone without betraying any symptoms of its formation. So good an observer as Lund (of Copenhagen) has seen abscess develop during his careful observation of a patient without his suspicion being aroused until after the abscess had developed. If this is so, so stormy a course as the usual thrombosis of the lateral sinus will almost certainly cover up the less obtrusive symptomatology of abscess formation; or if it is observable it is seen "through a cloud" as we have often remarked.

Confining our work to neurology we find it impossible to diagnosticate positively abscess of the brain in the presence of known thrombosis of the sinus; we must have the operative findings of the otologist step by step to eliminate successively extradural abscess and sinus thrombosis. Even then, as indicated above, non-suppurative encephalitis and other forms of "pseudo-abscess" must still be eliminated by time and prolonged observation.

As a general rule, encephalitis which precedes the formation of abscess is heralded by a sudden rise of temperature, severe headache, vomiting and focal symptoms. The pulse rate is usually rapid but may be slow because of the increased pressure incident to the inflammation and edema. These symptoms of the initial stage then subside and leave the characteristic picture of cerebral abscess (manifest stage).

One can generalize but little concerning symptoms of abscess of the brain. While it would seem theoretically an excellent approach to determine first whether an abscess is present in a given case this procedure as such is not very profitable, because the focal symptoms often overshadow the general ones and govern the clinical picture. The headache, vomiting, slow cerebration, bradycardia, mild choking of the disks and slight rigidity of the neck are all characteristic but not specific. To take up the symptoms systematically under the headings of (1) general, (2) general cerebral, (3) focal cerebral, (4) cranial nerve, (5) meningeal and (6) spinal fluid as is usually done would take us entirely too far afield. We shall confine ourselves to an enumeration of focal symptoms, as these occur from abscess in temporal, cerebellar, parietal, frontal and occipital locations. One must keep in mind that an abscess may even form on the side opposite the otitis.

In discussing symptoms of temporal abscess secondary to thrombosis of the lateral sinus, we do not overlook the fact that most temporal abscesses are formed by direct extension through the bone, and

hence are not intracranial complications of sinus thrombosis. However, otogenous abscess near the tip and far posteriorly in the temporal lobe probably arise by extension through the venous channels.

A. Symptoms Indicating Temporal Location.—When the major side is affected amnesic aphasia is extremely common. The patient is unable to name objects shown him though clearly aware of their identity. He frequently says, "I know but I can't say it." Only rarely does he fail to comprehend spoken words, but frequently he cannot write. Auditory hallucinations occur as do uncinate fits but these are not common. If one will take the trouble to test the visual fields and if the patient is sufficiently clear mentally, not infrequently hemianopic defects are found. Because of the difficulty in having the patient sit before a screen and in using a portable perimeter, the fields are generally not taken except by the usual rough tests. If the abscess is on the right side in a right handed individual, it may be nearly "silent." However, its very silence leads us to suspect its location. Conjugate deviation of the eyes may occur when the abscess is fully developed.

Reflex changes in the form of increase of the deep and diminution of the superficial ones on the contralateral side and pathological reflexes are common. Weakness of the contralateral limbs occurs later. Choking of the disks is not common in temporal abscess, though slight haziness of the margins is often seen.

B. Symptoms Indicating Cerebellar Location.—It might seem a simple matter to distinguish cerebellar abscess from temporal but the reports in the literature would rather seem to contradict this idea. In numerous cases of cerebellar abscess the surgeon has explored the temporal lobe before the cerebellum. This is due to the fact that the weakness of the affected limbs is about the same in both instances, changes in the fields of vision are often lacking in both, the early changes in the fundi are identical and the patient is usually too ill for the certain elicitation of the cerebellar ataxia. In addition to these factors the patient is too ill for vestibular tests to be done which would usually differentiate the two sites. Temporal location is about twice as common as cerebellar.

The helpful points in the differentiation are as follows: Cerebellar abscess is frequently, while temporal is rarely, developed through the labyrinthine route. The history is therefore of value. Operative findings may point the way of the infection when the mastoidectomy is done. The weakness resulting from cerebellar abscess is ipsilateral and never complete, while in temporal it is contralateral and becomes complete. (It is to be admitted, however, that it may be difficult to

determine which is the offending ear and hence the laterality of the lesion may be in question.) Choking of the disks is common (and early) in cerebellar and uncommon (and usually late) in temporal abscess.

The nystagmus, which may be present in either type of abscess, is usually slight in temporal and marked in cerebellar abscess. The meningeal symptoms are usually marked in cerebellar lesion and not in temporal. True adiadokokinesis and past pointing do not result from a temporal lesion but the weakness of the limbs may simulate it. The most important single fact is that, if vestibular tests are normal cerebellar abscess is excluded.

C. Symptons Indicating Parietal Location.—Abscess of the parietal lobe is prone to give rise to jacksonian convulsive seizures. This fact is extremely important because it is a common misconception that such seizures are strongly indicative of frontal location. Our experience has shown that jacksonian attacks are more common in parietal than in frontal abscess and also that parietal abscess is far more common than usually supposed. Conjugate deviation of the eyes and perhaps also the head is common in parietal abscess, and paralysis, first affecting the upper, then the lower extremity usually follows the convulsions. Another important symptom is rapid atrophy of the affected limbs as though the lesion were in the spinal cord. Hypoesthesia to all forms of sensation is common. Astereognosis, if present, is of great diagnostic value. Aphasic symptoms are ordinarily present in parietal abscess but these (because of the inflammatory zone which makes the lesion diffuse) do not differ essentially from those of temporal abscess.

Ideokinetic apraxia of the opposite limbs may result from a lesion of the posterior parietal lobe. In this condition the patient knows what he wishes to do but is unable to perform the intended act because he has forgotten how to make the movement. With sufficient strength and clarity of purpose, then, he cannot do what one asks of him.

D. Symptoms Indicating Frontal Location.—The frontal lobe is so large that one must not expect merely one but several frontal syndromes. Lesions of the anterior part tend to cause changes in personality and pathologic jocosity but these symptoms are not noticed unless the abscess becomes subacute or chronic. There is a strong tendency for large lesions of the frontal lobe to simulate lesions of the contralateral cerebellar hemisphere. The patient may develop nystagmus and a tendency to fall straight backward. Actual adiadokokinesis does not occur but cerebellar ataxia may be closely simu-

lated. Carphology, or reflex grasping and groping, is pathognomonic of a frontal lesion.

Lesions in the posterior part of the frontal lobe give totally different symptoms. Irritative lesions of the motor strip cause jacksonian seizures with conjugate deviation of the head and eyes and paralytic lesions cause paralysis of opposite limbs and of lateral gaze. If the lesion affects the motor strip itself, rapid atrophy occurs as in parietal lesions and kinetic apraxia of the limbs results, i. e., the patient, while able to use the forearm, does not have power of individual movement of the fingers. If the lesion is low down toward the operculum, aphasia of Broca appears. One must remember that accompanying encephalitis may add to this sensory aphasia.

Concerning the reflex changes in lesions of the frontal lobe, it is important to keep in mind that if the lesion is anterior to the motor strip spasticity results, but if it is in the motor strip itself flaccidity with increased deep reflexes occurs. Babinski sign will most likely be present in either case.

E. Symptoms Indicating Occipital Location.—The occipital site is the rarest of the lobar locations. This is because of its small size. At this point the superficial cerebral veins are shortest and the blood flow may go either toward the superior sagittal or the lateral sinus. When the abscess does locate here, homonymous hemianopsia is almost invariable. If the lesion is on the major side, the patient loses ability to read and sometimes also ability to identify colors or even objects. The faculty of naming them is lost first. Sometimes the patient loses himself in space and has no sense of direction at all. In general, lesions of the major occipital lobe cause much more psychic disturbance than do lesions of the frontal lobe, even when the major lobe is the one in question.

5. SUBDURAL ABSCESS

In a previous report on autopsy findings in 10,000 cases (1934) we cited three cases in which subdural abscess was associated with thrombosis of the lateral sinus. In fourteen other cases multiple intracranial lesions were observed. Subdural abscess occurs at times at a distance from the focus of infection. Rüedi (1930) has presented the general aspects of the subject.

Clinically, when subdural abscess is associated with extradural abscess, abscess of the brain itself or with meningitis, its symptomatology cannot be evaluated. When it occurs as a small focal lesion as at the tip of the petrous bone in the gasserian sheath, it may be suspected from the focal symptoms. When it occurs as the only essen-

tial lesion and is of any appreciable size, it gives focal symptoms closely resembling those of cerebral abscess. From a study of the literature we believe that subdural abscess has at times been drained and reported as a brain abscess. Macewen's (1893) most excellent case was carefully differentiated and is an outstanding example of this condition.

6. SPINAL FLUID FINDINGS

In thrombosis of the venous sinuses there is usually some degree of meningeal reaction and this determines the fact that the spinal fluid shows an increase in cells and globulin. At first these cells are polymorphonuclears, and, as the reaction subsides, they become predominantly lymphocytes. The spinal fluid findings will not be discussed in detail. Suffice it to say, there are no absolute determinants of meningitis except a positive culture on the fluid. All other findings in the fluid may occur with only a reactive meningitis.

In encephalitis there is a definite polymorphonuclear increase which may reach into hundreds per cmm. The same is true of the encephalitic stage of abscess of the brain which is essentially the same condition. The absolute count decreases and the character changes from polymorphonuclear to lymphocyte predominance as an abscess encapsulates. The lymphocyte count then decreases until, during the stage of latency, the cell count is again normal. If the abscess now flares up, as it may do several times before it bursts, the cell count again increases. If this behavior is kept in mind, the knowledge is of great value in differential diagnosis from meningitis. In subdural abscess, the spinal fluid findings are the same as in cerebral abscess.

The globulin content and other chemical constituents have been studied in detail by many men but so much depends upon the technic and individual factors that the methods are not yet of much practical value.

CONCLUSIONS

1. The intracranial complications of otogenous thrombosis of the lateral sinus are usually due to venous obstruction or to retrograde extension of infection into the afferent vessels and from thence into the meninges or brain. The anatomic arrangement of the intracranial venous system accounts largely for the distribution and character of many of these lesions.

2. The intracranial lesions which may follow thrombosis of the lateral sinus may be benign and transitory (local edema of meninges and brain; serous or reactive meningitis) or malignant and often lethal (subdural abscess or hemorrhage, septic meningitis, extension

into other venous channels with red softening (non-suppurative encephalitis) or abscess formation.

3. All focal lesions (edema, red softening, subdural or encephalic abscess) may be found in almost any situation in the intracranial space due to the communications in various parts of the venous system.

4. In most of the fatal cases of otogenous thrombosis of the lateral sinus some other intracranial lesion is found at autopsy. These lesions may be coincidental, developing as a result of extension along some other path from the middle ear, or consequential, due directly to the thrombus in the lateral sinus.

5. Transitory cerebral or cerebellar symptoms result from stasis in the local veins (local edema of cortex or meningitis) or to non-infected thrombosis (red softening) or infected thrombosis (septic meningitis, subdural or encephalic abscess) of these veins.

6. Transitory symptoms may follow abrupt (operative) occlusion of the lateral sinus or jugular vein if there is no pre-existing thrombus in the sinus.

7. The character and location of localized meningeal and cerebral lesion is determined by a study of the neurologic symptoms and signs and a survey of the clinical course of the lesion.

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III

AN ANATOMICAL AND CLINICAL STUDY OF CENTRAL LESIONS PRODUCING PARALYSIS OF THE LARYNX

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Ann Arbor

One has only to reflect upon the developments in laryngology during the past twenty years to realize that few, if any, basic or significant discoveries have been made in the neurological phenomena of the larynx. The problems which were discussed two decades ago in the didactic and clinical presentations of my senior year in the medical school, are yet unsolved and those studies in research which have been enthusiastically announced in our medical publications from time to time have failed, with but few exceptions, to live beyond that feeble period of existence which we term the preliminary report. The precise functions of the various elements of the laryngeal nerves are still under debate. The histological variations of the laryngeal nerves and of the muscles of the larynx are matters of scientific conjecture. The component parts of the central mechanism, have not been identified to the complete satisfaction of scientists in this field. The physiologic reactions of the neuclear cells still remain a mystery, and the character of the impulses which, upon stimulation traverse the afferent and efferent fibers of the laryngeal nerves, is as obscure today as it was at the time when laryngologists first gave thought to a study of the activities of nervous tissue.

On the face of it then, and in spite of a few isolated examples of brilliant investigation, our record of achievements in laryngological research in the past twenty years is not an enviable one. There are innumerable studies concerning which we are much in the dark and many problems whose solutions will make desirable contributions to the science of laryngology. I emphasize this fact to show the difficulties one encounters in assembling knowledge of laryngeal paralysis which is not a matter of historical record. Actually, however, we have a comprehensive grasp of the clinical manifestations of laryngeal paralysis, but this has not been acquired within the past two decades. The pioneers of laryngology designated the etiological factors, described the symptomatology and established principles of treatment, which for the most part do not require revision or modification as the result of any modern discoveries of basic significance

in this field. All of which again emphasizes my point of view that any effort on my part to correlate and publish our present day clinical knowledge of laryngeal paralysis would result merely in a repetition of medical history. The subject might lend itself admirably to embellishments and glorification but the product remains unaltered and the method open to censor, and properly too, by those who hold priority rights to the original publications.

It is not my intention, therefore, to give a didactic discourse on paralysis of the laryngeal nerves. I need not point out to you the reasons for laryngeal paralysis following operations upon the thyroid gland. I need not take your time to tell you that mitral stenosis with its consequent dilatation of the left auricle may cause paralysis of the left inferior laryngeal nerve, that mediastinal tumors, tuberculous fibrosis of the left lung and suppurative processes in the cervical region may produce a similar effect. It would be presumptuous on my part to describe to you the appearance of the larynx in the various forms of laryngeal paralysis. These are clinical facts exhaustively treated in our medical text books and well known to all of you.

There are, in any scientific discussion, many acceptable methods of presenting a subject but one which invariably attracts my interest is the plan of combining an anatomical study with the clinical manifestations of disease. I believe that we frequently lose the vital concepts of a disease entity by our failure to correlate what we know anatomically with what we observe clinically. Any medical problem backed by indisputable anatomical facts and supported by clinical example is, in my opinion, more clearly understood, more logically classified and better accredited by those who have given thought to it.

This method with its apparent advantage seemed particularly adaptable to a study of laryngeal paralysis. For obvious reasons I shall not attempt to apply it to lesions affecting the peripheral nerve trunks. As previously stated the peripheral pathways of the laryngeal nerves and the location and character of the morbid processes which affect them are so well recognized and so admirably presented in our text books and medical literature that further reiteration on these points would only serve to squander your time. However, in the matter of the intracranial distribution of the fibers of the laryngeal nerves there is much to be learned and many pertinent facts to be acquired by the plan of study above described. In this connection I have attempted to outline in a general way the applied anatomy of the laryngeal nerves within the central nervous system and to designate the possible locations of lesions affecting the nerve fibers along

their entire course from their cortical cells of origin to their sites of exit from the brain stem. In most instances I am privileged to illustrate my points by clinical records and by pathological sections obtained at autopsy. Needless to say a study of this character entails something more than a mere consideration of the laryngeal nerves. Central lesions are prone to involve neighboring structures and to produce neurological syndromes so that a recognition of associated paralysis is essential to a study of this kind. I will therefore direct your attention to the various neurological aspects of the lesions under discussion keeping as it were the laryngeal nerves predominantly in the picture and giving consideration only to those nerve disturbances which with those of the laryngeals comprise a neurological complex.

ORIGIN, COURSE AND DISTRIBUTION OF CORTICO-BULBAR FIBERS

Cortico-bulbar fibers carrying motor impulses to the lower motor neurons which supply the larynx have their cells of origin in the Betz cells or giant pyramidal cells in the lower part of the precentral gyrus of the cerebral cortex. The neuraxes of these cells pass as medullated fibers through the genu region of the internal capsule, enter the cerebral peduncle with other corticobulbar and corticospinal fibers in the middle three-fourths of that fiber mass, and below the peduncle interdigitate with the fibers of the pons. In the pons part of the bundles swing dorsalward with other corticobulbar fibers as the pontine division of the aberrant pyramidal tract and continue caudalward in close approximation to the medial lemniscus system. Below the pons the corticobulbar fibers for the laryngeal group lie partly within the pyramids of the medulla and partly as aberrant pyramidal fibers intermingled with the ventral portion of the medial lemniscus systems. They terminate, after a partial decussation, in the middle portion of the nucleus ambiguus around the cells of origin for the superior laryngeal nerve and in the caudal part of the nucleus ambiguus around cells of origin of the recurrent nerve. Thus the impulses brought to the cells of origin of the recurrent nerve on the left side will arise from both the left and right precentral regions of the cerebral cortex and this will be true whether the fibers course in the aberrant pyramidal system or in the pyramids.

The above anatomical facts have an important clinical application in that lesions above the nucleus of origin of the laryngeal nerves (n. ambiguus) can produce a paralysis of the larynx only when they are large enough to affect the corticobulbar fibers on both sides in which event the paralysis is bilateral and of the spastic type. The decussation of the corticobulbar fibers renders supranuclear lesions incapable of producing a unilateral laryngeal paralysis. Lesions with-

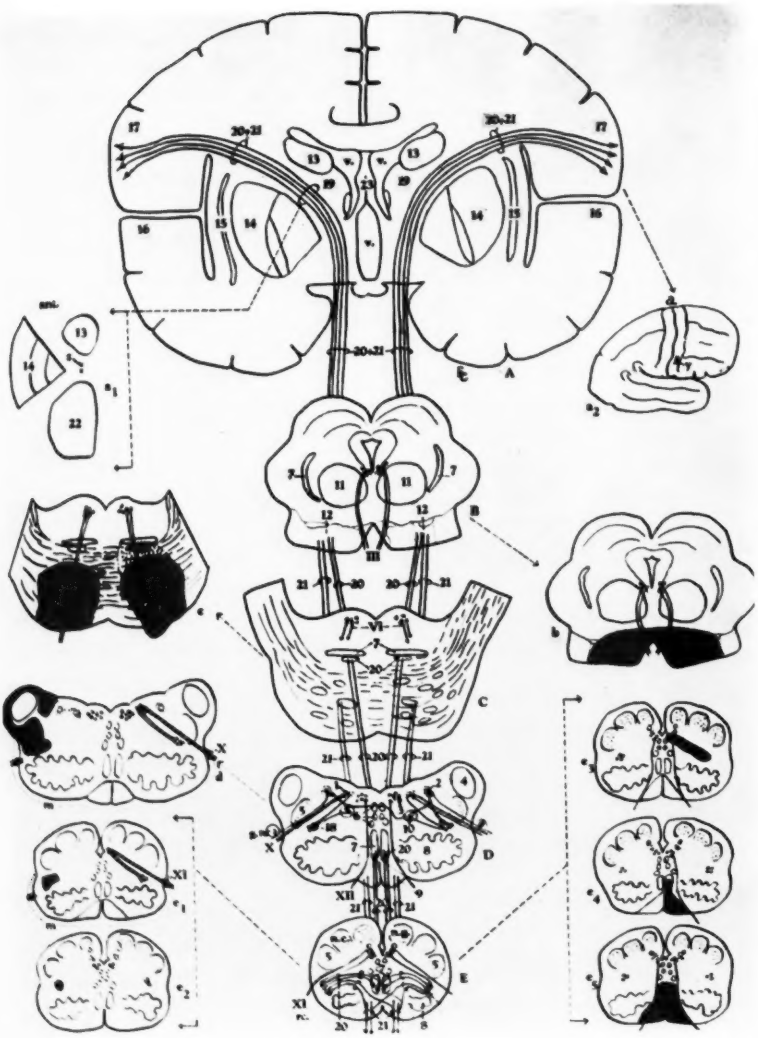

Fig. 1. Diagram showing origin, course and distribution of corticobulbar fibers carrying motor impulses to the lower motor neurons which supply the larynx. On both sides of the main diagram the localization of representative lesions, some of which produce laryngeal paralysis, is presented.

1. Dorsal efferent nucleus (of Xth nerve in figure labeled).
2. Fasciculus solitarius and associated nuclear gray.
3. Motor nucleus of XIIth nerve.
4. Restiform body.
5. Nucleus of the descending root of V.
6. From above downward the tracts are: medial longitudinal fasciculus, medial tecto-spinal, ventral spino-thalamic (in this approximate position).
7. Medial lemniscus (In the pons and midbrain levels this bundle includes, in addition to the medial lemniscus proper for proprioceptive and 2-point tactile discrimination, also the associated ventral spino-thalamic for tactile from the body, the lateral spino-thalamic for pain and temperature from the body, and the ventral secondary ascending tract of V, which carries pain and temperature, with some general tactile, from the head).
8. Inferior olivary nucleus.
9. Pyramid (note emerging XII nerve at side).
10. Medial reticular nuclear gray.
11. Red nucleus.
12. Position of cortico-(bulbo)-spinal and certain associated aberrant pyramid fibers in midbrain; lateral cortico-bulbar (an aberrant pyramidal system) overrides it.
13. Caudate nucleus.
14. Lentiform nucleus (putamen portion).
15. Claustrum.
16. Superior temporal gyrus (temporal operculum of island).
17. Motor area of cortex.
18. Nucleus ambiguus.
19. Dorsal thalamus (in transverse plane).
20. Aberrant pyramidal tract.
21. Cortico-(bulbo)- spinal tract.

20 and 21. (See numbers above.)

22. Dorsal thalamus (horizontal plane).
23. Columns of fornix.

in the nucleus ambiguus or infranuclear in location will produce a flaccid type of paralysis unilateral in character if one side alone is involved and bilateral only when both nuclei or both infranuclear pathways are affected.

It is frequently stated in our laryngological texts that central lesions usually produce a bilateral paralysis of the larynx. While this is probably true I am convinced that unilateral paralysis of central origin is not an uncommon condition. When both sides of the larynx are paralyzed, phonation and respiration are seriously disturbed and the condition of the patient becomes so grave that the laryngologist is promptly consulted. In the case of a unilateral paralysis of the larynx, there is often but slight impairment of laryngeal function and because the associated neurological disturbances dominate the picture the slight alteration of voice which may be present is considered too trivial to merit investigation. A routine study of many neurological cases over a period of twenty years has brought to my attention many examples of unilateral laryngeal paralysis and only a few in which the central disturbance involved the laryngeal nerves on both sides.

The following pages deal with the localization of representative lesions in the central nervous system and the neurological signs and symptoms which they produce. Insofar as possible actual cases are cited in support of the theoretical examples of lesions presented in the small diagrams. See Fig. 1.

DESCRIPTION OF LESIONS ILLUSTRATED

Large hemorrhages, tumors, abscesses or other lesions involving the cortical laryngeal areas on one side only (in position indicated in Fig. 2) will not produce paralysis of the laryngeal muscles because the corticobulbar path to the nucleus ambiguus (which gives rise to the motor fibers of the superior laryngeal and vicunens nerves along with other motor fibers) are both crossed and uncrossed. Destruction on both sides of the cortex at the appropriate regions would produce a bilateral laryngeal paralysis of the spastic type, but such lesions are exceedingly rare. (It is to be remembered that supranuclear lesions produce a spastic paralysis while lesions within the nucleus or infranuclear in type produce a flaccid paralysis.)

Lesions of the internal capsule (frequently hemorrhages) in the germ region on one side do not produce laryngeal paralysis for the reasons cited above. The position of the path is indicated in Fig. 3.

A lesion such as that indicated in Fig. 4, which is often syphilitic in character, may involve the brain in the region of the interpeduncu-

Fig. 2 (Sec. a in Fig. 1). Horizontal section of brain showing swelling and destruction of the right frontal lobe due to a large abscess. Although this lesion produced a motor aphasia, Jacksonian convulsions and paralyses of the opposite side of the body, it did not cause a paralysis of the larynx for the obvious anatomical reasons above described.

lar fossa. If such a lesion extends far enough lateralward to involve about four-fifths of the basis pedunculi a spastic paralysis of the muscles of the body and head (except possibly some eye muscles) and larynx will result. The emerging fibers of the oculomotor nerve will be involved, and a lower motor paralysis of the muscles supplied by this nerve will be observed. Whether or not a spastic paralysis will be observed in the other eye muscles will depend upon the extent of the lesion.

A lesion (frequently syphilitic in character) may involve the paths in the basal part of the pons. The regions mapped in solid black on both sides of Fig. 5 show two circumscribed lesions of this type which would result in a lower motor neuron paralysis of both

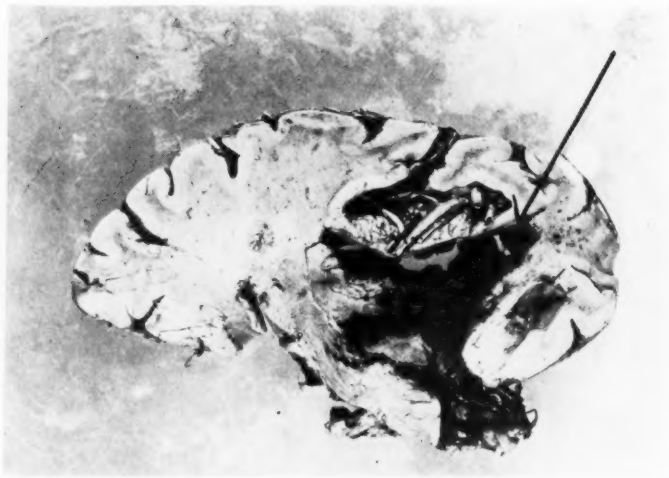

Fig. 3. (Sec. a1 in Fig. 1). Section of the brain through the internal capsule and the thalamus showing extensive damage to the insula and thalamus due to recent arterio-sclerotic hemorrhage. Although the lesion was most extensive, producing coma, contra-lateral hemiplegia and hemianesthesia, it did not produce a laryngeal paralysis for the obvious anatomical reasons above described.

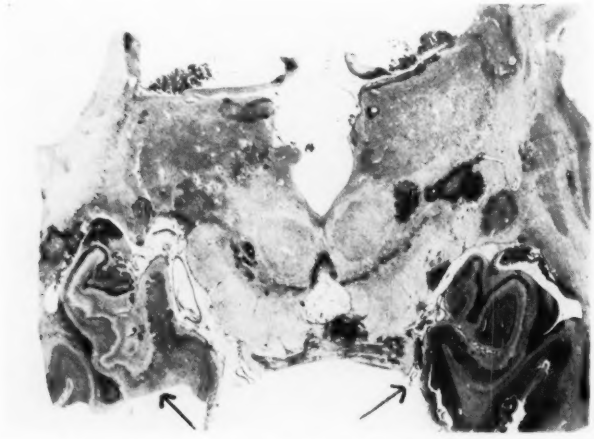

Fig. 4 (Sec. b in Fig. 1) Section through the pulvinar, the mid-brain and the basilar part of the temporal lobe showing multiple lesions of acute multiple sclerosis. The neurological findings were hemi-ballismus, Parkinsonian movements, impairment of the associated upward movements of the eyes and bi-lateral laryngeal paralysis of the upper neurone type.

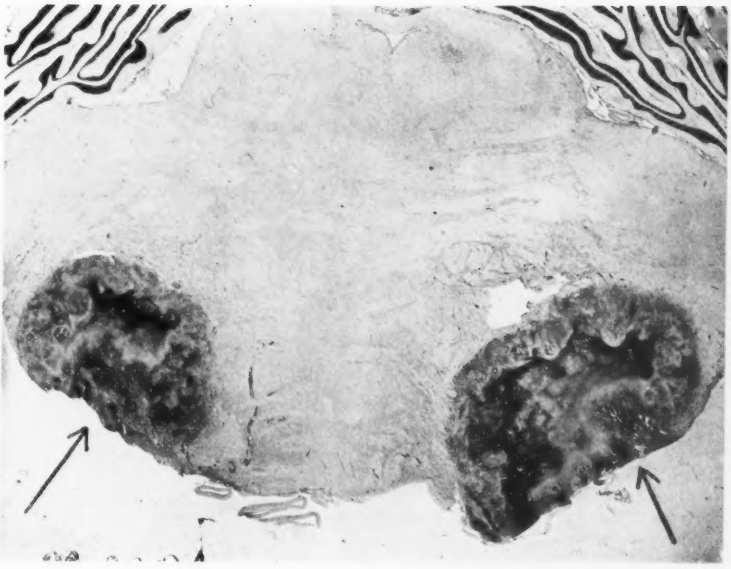

Fig. 5 (Sec. c in Fig. 1). Section through pons showing two large gummata in its base. The neurological findings were as follows: Lower motor neuron paralysis of both lateral recti muscles and a paresis of all extremities with an associated ataxia. Even in the presence of such widespread paralyses, the larynx was not affected for the anatomical reasons above described.

lateral recti muscles (supplied by VI nerve), and a spastic paralysis of both sides of the body. The larynx, however, would not be involved. If the area of destruction is increased still further as indicated by the regions enclosed within the dotted line in Fig. c, the aberrant pyramidal system may be involved on one side, but there is still a possible supply to the right nucleus ambiguus and left nucleus ambiguus through the intact aberrant tract of the other side. A unilateral laryngeal paralysis of the spastic type, in addition to other paralyses will occur if both aberrant tracts are involved in this lesion (if the medial lemniscus is involved proprioceptive and tactile disturbances of the trunk and extremities will appear).

Fig. 6 illustrated a lesion which frequently occurs as a result of thrombosis of the posterior inferior cerebellar artery. A nystagmus would develop because of involvement of the vestibular area just medial to the inferior cerebellar peduncle. Interference with the descending root of the V nerve and its nucleus would lead to a loss

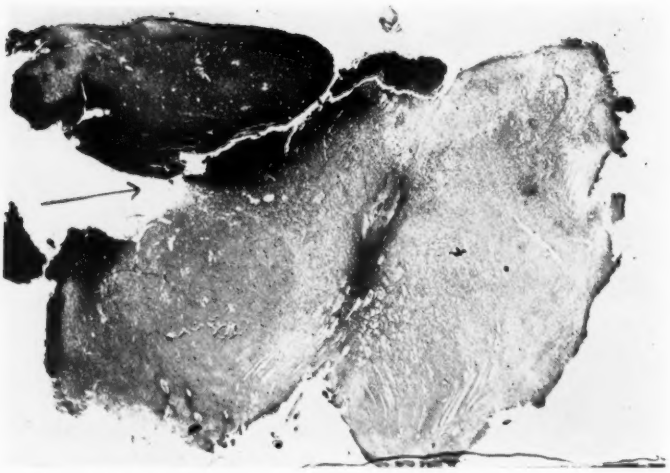

Fig. 6 (Sec. d in Fig. 1). Section through the mid-brain with extensive destruction due to arteriosclerotic hemorrhage. The patient was comatose with contra-lateral hemiplegia, hemianesthesia and hemianopsia of the opposite side. Bilateral loss of visual reflexes and interference with ocular movements. This section does not fit into Fig. d of the anatomical chart because it is at a higher level. Nevertheless, it shows the effect of hemorrhage and such a pathological change may occur lower down in the medulla destroying the nucleus ambiguus and producing a unilateral laryngeal paralysis.

of sensibility (particularly pain and temperature, and possibly some tactile) from the part of the face supplied by the ophthalmic branch of the fifth nerve with increase to the maxillomandibular as the lesion ascends. Interference with the lateral spinothalamic would produce a loss of pain and temperature (but not tactile) sensibility from the opposite side of the body. On account of the injury to the left inferior cerebellar peduncle, there would be difficulty in maintaining equilibrium, the patient showing a tendency to swing to the left side. Involvement of the nucleus ambiguus would lead to a lower motor neuron paralysis of the laryngeal muscles.

The letter m marking the dotted lines in Figs. d and e_1 of Fig. 1 indicates the spread of a meningitis about the pons which would involve the nerve roots of both the X and the XI nerves and produce in addition to other paralyses, a paralysis of the laryngeal muscles. This, no doubt, is the common pathological change responsible for bilateral laryngeal paralysis in patients suffering from a syphilitic meningitis.

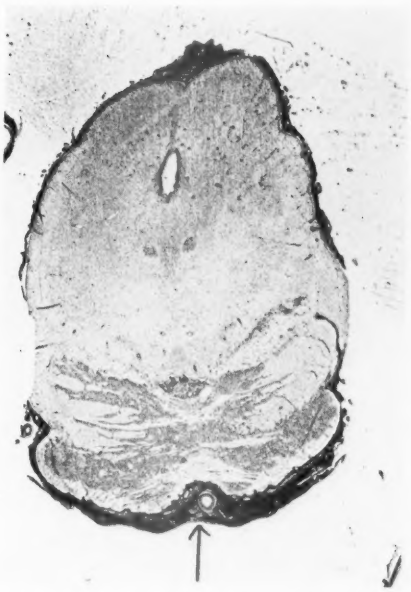

Fig. 7 (m in Sec. d and e in Fig. 1). Section through upper part of pons and mid-brain showing an acute purulent meningitis with extensive infiltration of the meninges. The clinical findings were diffuse headache, fever, cervical stiffness and pain, Kernig's and Brudzinski's signs and paralysis of both laryngeal nerves.

Fig. 9 shows a lesion of the left lateral spinothalamic tract and the left nucleus ambiguus. It is occasionally the result of hemorrhage in the medulla oblongata. Such a lesion produces a lower motor neuron paralysis of muscles supplied by the left recurrent laryngeal nerve and the left vagus. The destruction of the lateral spinothalamic tract, resulting from this lesion, would also produce a loss of pain and temperature on the opposite or right side of the body. (Syndrome of Avellis.)

Fig. e_2 (in Fig 1) illustrates a lesion of the left nucleus ambiguus producing a paralysis of the muscles of the larynx on the affected side (other striated muscles supplied from this nucleus through the IX and X nerves may also be affected if the lesion is large enough). The nucleus ambiguus consists of a long band of motor neurons, the most cephalic of which supply fibers through the IX nerve to the stylopharyngeus muscle. Next caudalward lie the cells supplying to the vagus and the superior laryngeal nerve. The caudal end of the nucleus ambiguus supplies fibers through the

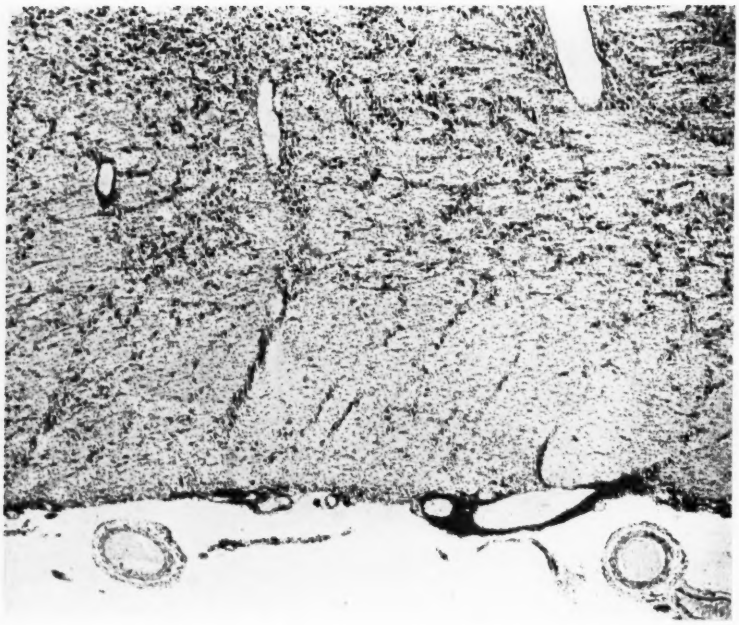

Fig. 8 (m in Sec. d and e in Fig. 1). High magnification of exudate of syphilitic meningitis about the pons and medulla. This is the common pathological change responsible for bilateral laryngeal paralysis of central origin.

internal ramus of XI which joins X in the region of the jugular foramen and passes out with it to split off as the vecunens nerve. Within this caudal end of the nucleus ambiguus (as in other portions of the nuclear mass) the neurons are in groups. There is scientific reason for the opinion that this grouping is in accordance with adductor and abductor muscle function of the vocal cords and that the fibers arising from the nucleus retain this grouping in their course to the laryngeal muscles. (Russell.) A lesion of this character may be produced by a tuberculoma. Occasionally the accessory nucleus of the cord is likewise affected by such a pathological change producing a paralysis of certain muscles of the neck. (Syndrome of Schmidt.) A pathological section of this lesion is not presented, but if available would be similar to Fig. 9, except of smaller proportions.

Fig. 10 shows a lesion involving the right nucleus ambiguus and the right hypoglossal nucleus. Such a lesion (which is occasionally syphilitic) produces a lower motor neuron paralysis of the muscles supplied by the right vecunen, and (if large enough) by

Fig. 9 (Sec. E1 in Fig. 1). Section of medulla at the level of the decussation of the pyramidal tracts showing an irregular degeneration of the white matter. Lesion is within the area circumscribed by the dotted line (not particularly satisfactory for reproduction). The neurological findings were a unilateral laryngeal paralysis because of destruction of the nucleus ambiguus on one side and a loss of pain and temperature on the opposite side of the body. (Syndrome of Avellis.) This is a classical example of unilateral laryngeal paralysis of central origin.

the right laryngeal. Associated with the disturbed function of these nerves one may find paralyses of other striated muscles supplied by the vagus and the stylopharyngeal muscle supplied by the glossopharyngeal nerve. There will be a lower motor neuron paralysis of the muscles of the tongue supplied by the right hypoglossal nerve, so that the tongue is drawn to the left side. (Syndrome of Jackson.)

Fig. 11 shows a lesion (produced for example by thrombosis or hemorrhage of the anterior spinal artery) involving the right hypoglossal nerve, the right medial lemniscus, the right aberrant pyramidal tract and the right pyramid. In this type of lesion one will observe a disturbance in proprioceptive impulses and two point discrimination from the right side of the body. The patient will present a spastic paralysis of the muscles of the left side of the body and lower motor neuron paralysis of the right hypoglossal nerve. A paralysis of the laryngeal muscles will not be observed unless the lesion is so large that it involves all crossed and uncrossed cortico-

Fig. 10 (Sec. E2 in Fig. 1). Section of medulla showing extensive degeneration of white matter which destroyed the nucleus ambiguus and the hypoglossal nucleus on one side. The neurological findings were paralysis of the right side of the larynx and of the stylo-pharyngeal muscle together with a lower motor neurone paralysis of the muscles of the tongue supplied by the right hypoglossal nerve. (Syndrome of Jackson.)

bulbar fibers supplying the motor neurons of nucleus ambiguus, which neurons distribute through laryngeal branches (including those in the aberrant tracts and those with the pyramids). The absence of laryngeal paralysis is due to the fact that the intact pyramid and intact aberrant pyramidal system on the left side carry fibers which cross to the right nucleus ambiguus as well as uncrossed fibers to the left nucleus ambiguus. It is apparent, therefore, that an injury on the one side will not produce a laryngeal paralysis. If the lesion spreads dorsalward beyond the limits shown in the figure, there will be a loss of more tactile sensibility on the side of the lesion. (Syndrome of Babinski-Nageotte.)

Fig. 12. The lesion in this figure involves both pyramids, both hypoglossal roots and both aberrant pyramidal fibers (supplying fibers to the nucleus ambiguus). Such a lesion produces a bilateral spastic paralysis of the body muscles, a bilateral paralysis of the laryngeal muscles and a lower motor paralysis of the muscles of the tongue.

SUMMARY AND COMMENT

1. The course of the corticobulbar fibers carrying motor impulses to the lower motor neurons, which supply the larynx, is outlined. Particular attention is called to the pontine division of the aberrant pyramidal tract and the partial decussation of the corticobulbar fibers for the laryngeal group before they terminate in the

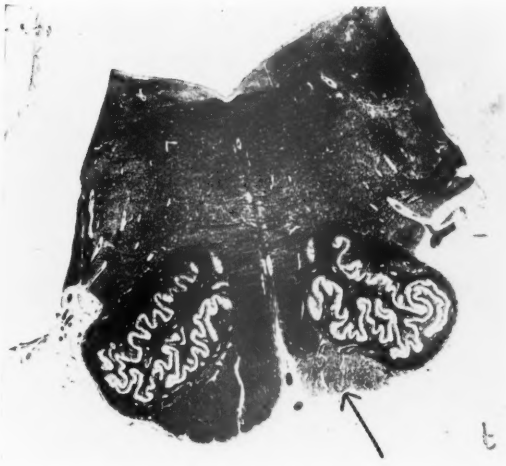

Fig. 11 (Sec. E4 in Fig. 1). Section of medulla showing unilateral degeneration of the right pyramidal tract. The neurological findings were spastic hemiplegia on the left side of the body with an increase in muscle tone and reflexes as well as a positive Babinski on the left. A lower motor neuron paralysis of the right hypoglossal nerve was present. A laryngeal paralysis was not observed for the obvious anatomical reasons above described.

nucleus ambiguus. Therefore, the impulses brought to the recurrent nerves will arise in both the right and left precentral regions of the cerebrum; an anatomical fact of much importance when endeavoring to localize lesions causing laryngeal paralysis of central origin.

2. Lesions above the nucleus of origin of the laryngeal nerves, (n. ambiguus) cannot produce a unilateral paralysis of the larynx on account of the decussation of the corticobulbar fibers. When large enough to involve the corticobulbar fibers on both sides, a bilateral laryngeal paralysis of a spastic type will be observed. Attention is called to a clinical differentiation seldom made by the clinician, namely, the spastic versus the flaccid type of laryngeal paralysis. Supranuclear lesions produce the former; nuclear and infranuclear lesions, the latter; and the careful observer will not fail to recognize these types when looking at the reflected image of the larynx in a mirror.

3. Unilateral laryngeal paralysis of central origin is not an uncommon condition. It is seldom observed, however, because it is usually associated with a preponderance of obvious neurological find-

Fig. 12 (Sec. E5 in Fig. 1). Section through medulla showing complete degeneration of both pyramidal tracts. The neurological findings were spastic quadriplegia with increase in muscle tone and reflexes as well as a positive Babinski on both sides. There was a bilateral paralysis of the laryngeal muscles and a lower motor paralysis of the muscles of the tongue.

ings which invite study and minimize the attention given to the special organs of the body. Likewise, in many cases of unilateral laryngeal paralysis the symptoms are so trivial that neither the patient nor the doctor is aware of their presence.

4. A description of hypothetical lesions in the cerebrum and brain stem is given. These are taken from the clinical study of a large series of neurological patients and supported wholly or in part by pathological specimens obtained at autopsy.

My gratitude is due Dr. Elizabeth Crosby for her kindness in contributing generously to the anatomical studies herein contained. I am thankful, too, for the valuable assistance of Dr. Konstantin Scharenberg in reviewing pathological material and aiding me in assembling the sections which appear in this manuscript.

UNIVERSITY HOSPITAL.

IV

AUDIOMETRIC STUDIES ON SCHOOL CHILDREN

III. Variations in the Auditory Acuity of 543 School Children Re-Examined After an Average Interval of Three Years*

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INTRODUCTION

The audiometer has been in fairly common use for a number of years, yet there are no systematic studies of the hearing variations beween two or more tests made on the same individual.[1] Since this is a question of great moment, one of the principal aspects of the investigation on the hearing of school children undertaken by the Office of Child Hygiene of the U. S. Public Health Service[2] is that of obtaining repeated periodical otolaryngologic and audiometric examinations on school children. This paper contains a statistical analysis of the first series of repeated tests and has for its purpose the presentation of certain characteristics in the variations of auditory acuity.

MATERIAL

In 1931, when the Office of Child Hygiene began this investigation of the hearing of Washington, D. C., school children, the hearing of almost 14,000 children was tested with a 4-A audiometer.[3] About 1,400 of these children, selected so as to represent equally children with good and with impaired hearing, were tested for air and bone conduction acuity with a Western Electric Company 2-A audiometer.[4] These children also received a fairly complete otolaryngologic examination. After a mean interval of almost three years, 543 of these children were retested by the same examiner, with the same instrument and under the same general environmental conditions. The hearing records of these 543 children constitute the material of the study presented in this paper. The selection of the children to be re-examined was entirely at random: as many as possible of those formerly examined were taken, regardless of previous auditory condition, age or sex. The ages of these children ranged from 8 to 17 years, with a mean age of almost 11 years.

*From the Office of Child Hygiene, U. S. Public Health Service.

CHANGES IN HEARING ACUITY BY AIR CONDUCTION

For each tone in the range of the audiometer and for each ear, the difference between the threshold at the first examination and that at the second examination has been computed. If the hearing acuity increased, the difference is preceded by a minus sign; if the hearing acuity decreased the difference is preceded by a plus sign. Zero indicates that there was no difference between the two tests in hearing acuity for the particular tone.

A graphic representation of the frequency distributions of the differences between the two tests for all the ears is shown in Fig. 1. In table I are given some statistical constants of these distributions. The sign and value of the arithmetical mean indicate the direction and degree of the hearing change, the standard deviation measures the variation, as does also the value of the percentage of ears with differences within plus and minus 5 decibels (db). In Fig. 1, broken lines connect the limits that for each tone are determined by one standard deviation above and below the mean difference.

From the table and figure the following facts are worthy of note:

1. On the average, the differences tend to cluster around zero, but there is a tendency towards a decrease in hearing acuity at the second test. However, the only averages which probably diverge significantly from zero are those for the tones of 64, 512 and 1,024 cycles. The values of these means are slightly more than three times their standard errors.

2. The variation in the responses to the two tests is least for the tone of 512 cycles and increases regularly for the lower and higher pitches. The maximum variation is found for the highest tone, 8,192 cycles. The values of the percentage of ears that for each tone show a difference equal to or less than 5 db. follow the same trend as that of the standard deviation.

From the values of the standard deviation it appears that at a second examination, for tones up to 2,048 cycles, inclusive, fluctuations of around 5 db. above or below the first threshold are found in the majority of cases. For tones of 4,096 cycles and above the fluctuations are definitely higher and approximate $\pm$ 10 db. Numerous hypotheses can be advanced to explain the differences between the degree of variation for each tone. It may be assumed that the response to tones of the conversational range is more consistent because these are tones to which the ear is more habituated; or that the morphologic structure of the ear is such as to favor greater variability

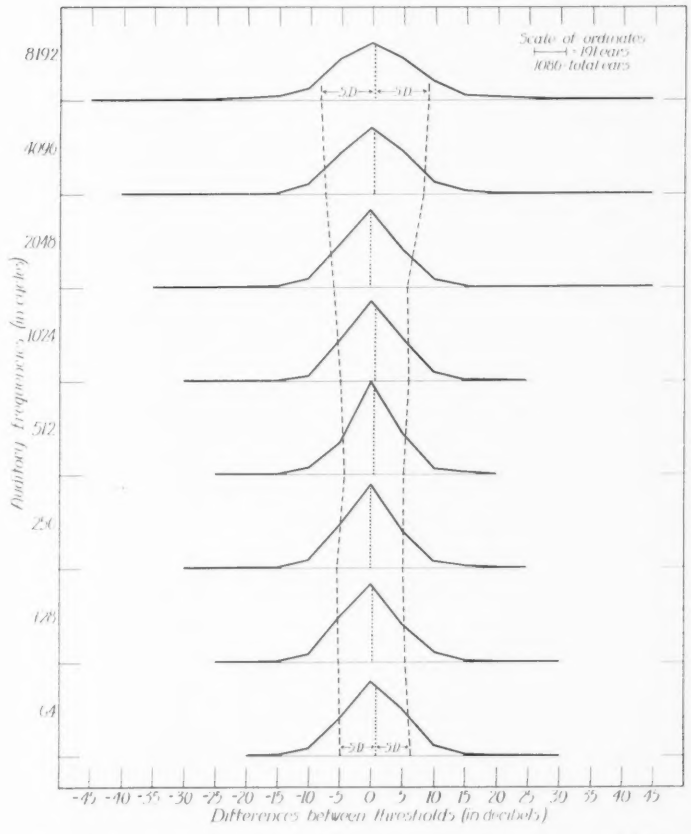

Fig. 1. Frequency curves of differences between auditory thresholds at first and second test. Both ears of 543 school children, 8 to 17 years of age, re-examined after an average interval of 3 years.

for the extreme high and low tones; or that this is caused by mechanical imperfections of the instrument. Any explanation, however, must await more facts.

CLASSIFICATION OF INDIVIDUAL CHANGES IN HEARING

In an investigation of hearing changes it is not sufficient to know the mode of variation of the single tones; it is essential also to obtain a synthetic view of how the individual ear varies for all the tones of the auditory range. In order to study this question, the following somewhat arbitrary procedure has been employed. For

each tone, the interval limited by one standard deviation above or below the mean (cf. Table I and Fig. 1) may be reasonably regarded as a range of "normal variation." A difference within these limits indicates, for the purpose of this study, no significant change in acuity; a difference outside these limits is regarded as an actual change in hearing. Since the threshold readings are given only in units of 5 db., the unit nearest the value of the standard deviation is employed as a limiting value. Thus, for the tones up to 2,048 cycles, inclusive, 5 db. is used in place of the standard deviation; for the tones of 4,096 and 8,192 cycles, 10 db. is similarly used. From Table I it is seen that within these limits the percentage of ears included varies from 86 in the case of 64 cycles to 94 for the tone of 4,096 cycles.

A change in hearing may occur for any tone or group of tones. In order to take this into account the auditory range is divided into low tones, 64 and 128 cycles; middle tones or conversational range, 256 to 2,048 cycles; high tones, 4,096 cycles and above. Improvement in hearing acuity, or the reverse, can be expected to occur for any of these tones or combinations thereof.

When each ear of this material is classified according to the definitions discussed above, the results shown in Table II are obtained. This table shows:

1. The hearing of exactly two-thirds of the ears remained unchanged. There were slightly more ears with decreased hearing acuity than with increased acuity.

2. When definite changes occurred, these involved tones of the conversational range more frequently than either the high or low tones.

3. In this material there were no cases in which the hearing acuity was increased for one group of tones and decreased for another.

The relative preponderance of changes involving the middle tones is easily understood, since these are the tones most affected by acute temporary diseases of the middle ear, the most common aural disturbances.

ASSOCIATION BETWEEN TONES IN RELATION TO CHANGES IN HEARING ACUITY. ASSOCIATION BETWEEN CHANGES IN RIGHT AND LEFT EAR

It is noteworthy that at the second test the hearing of two-thirds of the ears did not alter by more than 5 db. for tones up to 2,048 cycles and by 10 db. for tones of 4,096 and 8,192 cycles. From Table

TABLE I

Statistical constants of differences between auditory thresholds at first and second test. Both ears of 543 school children, 8 to, 17 years of age, re-examined after an average interval of three years.

	Auditory frequencies (in cycles)							
	64	128	256	512	1024	2048	4096	8192
Mean difference (in db)	.67 ± .12	.02 ± .11	—.02 ± .11	.47 ± .10	.53 ± .11	—.04 ± .12	.50 ± .16	.55 ± .18
Standard deviation (in db)	5.64 ± .08	5.28 ± .08	5.18 ± .07	4.75 ± .07	5.37 ± .08	6.05 ± .09	7.89 ± .11	8.56 ± .12
Per cent ears with differences equal to 0 ± 5 db.	86.28	88.58	89.96	91.62	90.42	87.29	81.22 (93.74)*	76.34 (91.99)*

* Per cent ears with differences equal to 0 ± 10 db.

TABLE II

Hearing changes in 543 school children, 8 to 17 years of age, re-examined after an average interval of three years.

Changes in Hearing Acuity	Number of ears	Per cent
Increased acuity		
All tones	7	.64
Low tones only	27	2.49
Low and middle tones	43	3.96
Middle tones only	52	4.79
Middle and high tones	11	1.01
High tones only	27	2.49
No change	724	66.67
Decreased acuity		
All tones	24	2.21
Low tones only	40	3.68
Low and middle tones	44	4.05
Middle tones only	47	4.33
High tones only	40	3.68
Total ears	1,086	100.00
Total increased acuity	167	15.38
No change	724	66.67
Total decreased acuity	195	17.95

I it is seen that there are from 86 to 94 per cent of the ears included within these limits. Therefore, if the variation in the response of the ear to each tone was independent of the variations in the responses to the other tones, 43 per cent of the ears would be expected to show, for all the tones, differences in thresholds within these limits. A fundamental theorem of the theory of probabilities states: The probability of the simultaneous occurrence of n independent events is equal to the product of the individual probabilities of the n events. Thus, for this problem: $.8624 \times .8858 \times .8996 \times .9162 \times .9042 \times .8729 \times .9374 \times .9199 = .4288$ as stated above. Actually, as has been mentioned, there are 66.67 ± 1.43 per cent of the ears that fall within the range specified. This indicates an association between the changes in acuity for one tone and changes in acuity for the other tones. The existence of such a relationship is also demonstrated by the fact that the hearing acuity of 2.85 per cent of the total ears increased or decreased for all the tones: 2.21 per cent decreased and .64 increased. The mathematical expectation, assuming that there is independence between the variations in the responses to each tone, is practically zero. (To be exact, the product of the individual probabilities is equal to 17 times 10^{-10}.) The inference to be

TABLE III

Actual and expected number of school children with specified type of hearing changes in right and left ear. School children 8 to 17 years of age re-examined after an average interval of three years.

Changes in Hearing Acuity of Right Ear.	Changes in Hearing Acuity of Left Ear						Total
	Increased		No Change		Decreased		
	Actual	Expected	Actual	Expected	Actual	Expected	
Increased	42	*13*	33	*51*	3	*14*	78
No change	42	*61*	282	*241*	47	*69*	371
Decreased	5	*15*	38	*61*	51	*18*	94
Total	89		353		101		543

drawn from these findings is that the greater or less variation in response to threshold stimuli is in part a function of the ear as a whole. That is, on the average, if the ear varies to a certain degree for one particular tone, it will also tend to vary in the same degree for other tones.

Not only does it appear that the ear tends to vary in the same degree and direction for all tones, but there is also a positive association between the variations of the two ears of the same individual. This is evident from Table III, which shows the actual and expected number of children whose hearing changed in one ear and did or did not change in the other. This tabulation demonstrates two things: (a) there are more than the expected number of children with a homologous hearing change in both ears; (b) there are less than the expected number of children in whom, at the second test, the hearing acuity of one ear was altered in a manner different from that of the other ear. The difference between actual and expected is especially marked for those children whose hearing improved in one ear while it became worse in the other. A chi-square value equal to 182 indicates that the hypothesis of independence between the ears does not hold. It can be asserted with confidence, therefore, that there is a certain degree of correlation between the variations of the right and of the left ear.

From these facts it is evident that in order to evaluate the significance of hearing changes, the effect of different therapeutic measures, and the importance of other factors, it is necessary first of all to determine the degree and extent of the individual variation. This is possible only by systematic analysis of a sufficient number of records which contain complete clinical information.

TABLE IV

Hearing changes in boys and girls, 8 to 17 years of age, re-examined after an average interval of three years.

Changes in Hearing Acuity	Boys		Girls	
	No. of Ears	Per Cent	No. of Ears	Per Cent
Increased acuity				
All tones	4	.78	3	.52
Low tones only	11	2.16	16	2.78
Low and middle tones	15	2.94	28	4.86
Middle tones only	21	4.12	30	
Middle and high tones	4	.78	7	
High tones only	18	3.53	9	
No change	338	66.28	387	
Decreased acuity				
All tones	12	2.35	12	2.08
Low tones only	17	3.34	23	3.99
Low and middle tones	20	3.92	24	4.17
Middle tones only	20	3.92	27	4.69
High tones only	30	5.88	10	1.74
Total ears	510	100.00	576	100.00
Total increased acuity	73	14.31	93	16.15
No change	338	66.28	387	67.18
Total decreased acuity	99	19.41	96	16.67

HEARING CHANGES IN BOYS AND GIRLS

In Table IV are presented the absolute and relative distributions of the types of changes found for the boys and girls. For the girls, the percentage of ears with no hearing change at the second test is very slightly and insignificantly lower than the same percentage for the boys. There are relatively more boys than girls with decreased hearing acuity and the reverse is true for increased acuity. When the changes are considered in relation to the tones involved, the only statistically significant difference between the boys and girls is in the incidence of decreased perception for high tones. The ears of the boys showing this condition are three times as numerous as those of the girls. This fact, which was first observed by Bunch and Raiford[5] on hospitalized patients and confirmed by Ciocco[6] on hospitalized subjects and on school children, is another link in the chain of evidence regarding the relatively greater susceptibility of the males to loss of perception for high tones.

All of these facts point to the importance of the study of the individual in investigations of hearing variations in general and of high tone loss in particular.

HEARING CHANGES IN RELATION TO AUDITORY ACUITY AT THE FIRST TEST

Of the 1,086 ears which constitute this material, 962, or 88.6 per cent, had good hearing at the first test; 47, or 4.3 per cent, had a high tone loss; 45, or 4.1 per cent, had impaired hearing for conversation, with reduced bone conduction; 32, or 3.0 per cent, had a conductive type of deafness (impairment for air conduction but with good bone conduction acuity.) The terms "good hearing," "high tone loss," etc., are here used as previously defined by the author. The changes in hearing noted for the ears with the four conditions described above are summarized in Table V, in which are presented also the mean thresholds at the first and at the second examination of each group of ears. From this table it is seen that the proportion of ears with no change in hearing is much higher for the ears with good hearing than for the remaining: about 72 per cent of the ears with good hearing showed no change in auditory acuity, while this same condition is found in only 23 per cent of ears which previously had high tone loss, in 33 per cent of those with impaired air and bone conduction acuity and in 25 per cent of the ears with a conductive type of deafness.

For the ears previously classified as having good hearing, the table shows that at the first examination the average hearing acuity of the ears whose hearing remained unchanged was practically the same as the original acuity of the ears whose hearing decreased. Instead, the ears which improved are those whose audiograms at the first examination were on the borderline between good and impaired hearing as defined. The ears whose hearing decreased have at present average thresholds, which indicate that about one-third of the ears have now some impairment for conversation. In other words, during the average interval of three years, about 5 per cent of the children with good hearing have acquired some hearing impairment.

For the ears with high tone loss at the first examination, the mean threshold for the tone of 2,048 cycles is significantly higher in the ears whose hearing remained unchanged than in the others. This is the only essential difference between the average thresholds, at the first examination, of the ears which changed and those that did not. The improvement in hearing is especially marked for the tone of 8,192 cycles. However, the average curve still conserves, at the tone of 4,096 cycles, the characteristics of a high tone loss. The ears that decreased do so equally for all tones and not only for the high tones.

TABLE V

Means of auditory thresholds (in db.) at first and second examination of 543 school children; an average interval of 3 years between examinations. Ears classified according to acuity at first test and according to hearing changes.

Auditory Frequencies (in cycles)	Changes in Hearing Acuity					
	Increased		No Change		Decreased	
	1st Test	2nd Test	1st Test	2nd Test	1st Test	2nd Test
	Good Hearing at First Test					
64	5.00 ± .25	—.04 ± .22	1.64 ± .09	2.12 ± .09	1.15 ± .27	6.88 ± .35
128	7.92 ± .24	2.46 ± .20	5.05 ± .09	4.81 ± .09	4.34 ± .25	9.34 ± .35
256	11.79 ± .23	6.88 ± .21	9.26 ± .08	9.00 ± .09	8.59 ± .26	13.19 ± .34
512	15.38 ± .21	11.38 ± .21	13.27 ± .08	13.57 ± .09	12.80 ± .22	17.30 ± .29
1024	11.59 ± .20	7.50 ± .24	9.65 ± .09	9.80 ± .09	8.45 ± .27	13.88 ± .36
2048	8.79 ± .24	3.42 ± .25	5.91 ± .10	5.68 ± .10	5.16 ± .27	10.13 ± .37
4096	8.13 ± .29	3.96 ± .31	5.09 ± .12	5.38 ± .12	5.10 ± .31	12.43 ± .62
8192	5.88 ± .36	1.75 ± .30	2.83 ± .13	3.37 ± .12	3.62 ± .35	10.69 ± .64
No. of ears	120		690		152	
	High Tone Loss at First Test					
64	4.75 ± .08	1.25 ± .04	3.64 ± .10	3.64 ± .08	3.75 ± .07	10.00 ± .11
128	7.25 ± .08	5.25 ± .05	5.45 ± .07	5.45 ± .08	7.50 ± .09	12.81 ± .12
256	11.00 ± .07	9.50 ± .06	10.00 ± .08	10.45 ± .07	11.25 ± .06	18.13 ± .13
512	15.75 ± .07	14.25 ± .06	14.55 ± .08	15.91 ± .07	15.31 ± .08	22.81 ± .14
1024	12.00 ± .13	11.00 ± .10	13.64 ± .14	14.55 ± .15	11.88 ± .09	21.88 ± .25
2048	10.50 ± .14	6.75 ± .12	18.18 ± .37	16.36 ± .36	13.75 ± .20	24.69 ± .31
4096	31.00 ± .21	12.50 ± .19	34.09 ± .25	34.09 ± .29	34.38 ± .25	41.88 ± .28
8192	29.75 ± .23	7.00 ± .10	29.09 ± .33	29.55 ± .37	29.38 ± .26	36.25 ± .33
No. of ears	20		11		16	

Air and Bone Conduction Reduced at First Test

64	12.86 ± 1.43	5.71 ± .82	28.67 ± 4.13	28.33 ± 4.17	11.88 ± 1.15	23.44 ± 1.45
128	16.43 ± 1.29	8.93 ± .78	31.00 ± 3.81	31.33 ± 3.90	16.25 ± 1.21	26.25 ± 1.45
256	22.86 ± 1.17	13.21 ± 1.00	37.00 ± 3.48	36.00 ± 3.52	21.25 ± 1.45	30.00 ± 1.30
512	24.29 ± 1.27	18.21 ± 1.11	41.33 ± 3.25	40.67 ± 3.32	27.50 ± 1.19	34.38 ± 1.19
1024	22.86 ± 1.59	18.21 ± 1.76	38.00 ± 3.76	37.67 ± 3.77	27.50 ± 2.02	34.69 ± 1.83
2048	20.36 ± 2.40	15.36 ± 2.65	37.34 ± 4.13	37.67 ± 4.14	27.81 ± 3.13	38.13 ± 2.98
4096	20.36 ± 2.56	15.00 ± 2.83	36.33 ± 4.61	35.67 ± 4.60	29.69 ± 3.08	38.44 ± 2.96
8192	16.79 ± 2.42	14.29 ± 2.95	34.33 ± 4.70	33.33 ± 4.50	24.06 ± 2.60	35.63 ± 2.71
No. of ears	14		15		16	

Conductive Deafness at First Test

64	26.54 ± 2.94	20.00 ± 3.20	33.75 ± 3.71	32.50 ± 4.09	28.18 ± 2.46	40.00 ± 3.58
128	30.38 ± 2.71	21.92 ± 2.88	35.63 ± 3.60	38.13 ± 3.81	35.45 ± 2.89	41.82 ± 3.28
256	36.15 ± 2.44	28.08 ± 2.97	42.50 ± 3.82	41.88 ± 3.95	42.27 ± 2.82	50.00 ± 3.13
512	40.00 ± 2.29	32.31 ± 2.83	47.50 ± 3.10	48.75 ± 2.97	47.73 ± 2.76	54.09 ± 2.63
1024	39.23 ± 2.46	26.54 ± 2.90	46.88 ± 3.57	47.50 ± 3.63	45.45 ± 2.86	53.64 ± 2.42
2048	39.62 ± 2.03	24.62 ± 3.10	45.00 ± 3.68	46.25 ± 3.85	43.18 ± 2.89	50.45 ± 3.02
4096	41.15 ± 2.55	30.00 ± 3.81	46.88 ± 3.26	46.25 ± 3.81	47.73 ± 2.54	53.64 ± 2.57
8192	38.08 ± 2.63	25.38 ± 3.62	45.63 ± 3.12	44.38 ± 3.55	47.73 ± 2.76	49.55 ± 2.14
No. of ears	13		8		11	

Ears which, at the first examination, had reduced air and bone conduction acuity, show the following characteristics:

(a) No change in hearing was observed for the ears which at the first test had, on the average, the greatest impairment.

(b) When there was improvement or decrease in hearing, this occurred equally for all tones. The present average hearing for the ears which improved is still below that of the ears with good hearing. The present average thresholds of the ears which decreased are practically equal to those of the ears which remained unchanged.

With regard to the ears with a conductive type of impairment, at the first test, the following facts are to be noted:

(a) The hearing acuity of the ears which improved was at the first examination better than for the other two groups. The improvement is on the average more marked for tones of 1,024 cycles and above than for the other tones. However, on the average these ears are well within the range of what has been defined as impaired hearing.

(b) The decrease in hearing is, on the average, practically the same for all the tones.

These findings may be summarized as follows:

1. Ears with impaired hearing show greater variability than those with good hearing.

2. Ears with impaired perception for high tones only, when this involves the tone of 2,048 cycles, are less variable on second examination than those in which this tone is not impaired. When there is improvement for the ears with high tone loss, this is found especially for the tone of 8,192 cycles. Decrease in hearing is equal for all tones.

3. Ears with impaired hearing by air and bone conduction fluctuate only as long as the hearing loss has not reached a threshold which on the average is between 30 and 40 db.

4. When there is a conductive type of hearing impairment, improvement is observed only for those ears with a hearing loss which does not exceed an average threshold of 30 to 40 db.

It is of particular interest to note that in cases of high tone loss the original average hearing was the same for the ears which did and did not change. The inference to be drawn from this is that not all cases of high tone loss are due to nerve and end organ degeneration.

In their study of the pathology of high tone loss, Crowe et al.[7] found some cases without sufficient pathology of the nerve and end organ to explain the loss of hearing. Therefore, it should be of importance to find a criterion by which to differentiate between the loss due to the above conditions of the inner ear and that due to other factors.

The above facts also suggest that for ears with reduced air and bone conduction there is a level of impairment at which the hearing will neither improve nor deteriorate further. For the ears with conductive deafness, instead, this level either does not exist or occurs when the hearing loss is greater than that found in this material. However, for both types of hearing impairment, apparently there cannot be an improvement in hearing after the hearing loss has progressed beyond a certain level. A precise determination of this level cannot be attempted here.

The number of cases, the interval between tests and the clinical information at hand are not adequate to permit further investigation of the other factors possibly associated with hearing variation. It is sufficient to say that for these children no relationship was observed between the degree and form of hearing changes and age, condition of tympanic membrane or condition of tonsils.

SUMMARY

A statistical analysis of the variations in the auditory acuity of 543 children, examined twice in a period of approximately three years with a Western Electric 2-A audiometer, shows:

1. The variation in the responses to the tests is least for the tone of 512 cycles and increases regularly for the lower and higher pitches. The maximum variation is found for the highest tone, 8,192 cycles.

2. For tones up to 2,048 cycles a fluctuation of 5 db. above or below the original thresholds is found in the greatest number of ears. For tones of 4,096 and 8,192 cycles a fluctuation of ± 10 db. is equally frequent.

3. The hearing of exactly two-thirds of the ears remained relatively unchanged—that is, varied within the range stated in 2. When definite hearing changes occurred, these involved tones of the conversational range more frequently than either the high or low tones.

4. On the average, if the ear varied to a certain degree for one particular tone, it will also tend to vary in the same degree for other tones.

5. There is a positive association between the hearing variations of the right and left ear.

6. The incidence of a definite decrease in the perception for high tones at the second test was significantly higher in boys than in girls.

7. Ears with impaired hearing showed greater variability than those with good hearing.

8. Ears with impaired perception for high tones only, when this involved the tone of 2,048 cycles, were less variable on the second examination than those in which this tone was not impaired.

9. Ears with impaired hearing by air and bone conduction did not fluctuate after the hearing loss had reached a threshold which on the average was between 30 and 40 db.

10. When there was a conductive type of hearing impairment, improvement was observed only for those ears with hearing which did not exceed an average threshold of 30 to 40 db. for all tones.

The author wishes to acknowledge the valuable assistance of B. L. Jarmon, M. D., who made the audiometric and clinical examinations.

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V

HEADACHE FROM THE NASAL WALL*

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The cause of headache has been the subject of considerable speculation and controversy. Any line of investigation, therefore, is welcome, which leads to a better understanding of even a small part of this complex problem. This communication deals with one of the commonest headaches encountered, namely, a headache of nasal origin involving one or more branches of the ophthalmic division of the trigeminus nerve. The most interesting feature is the fact that in many instances the headache arises from the anterior ethmoid nerve (a branch of the trigeminus) on the lateral nasal wall, and in most cases no sinus disease is present nor any of the usual signs of nasal involvement. In appearance, the nasal mucosa is frequently quite normal. It is not until the test, which is described under "examination" in the following description, has proved positive that the source of this headache is found to be in this area.

On obtaining the history and observing the course of a typical case, it is evident that the usual explanations of nasal headache are inadequate. In accepting the hypothesis which will be advanced in this paper, considerable doubt is cast on the generally accepted theory respecting the cause of sinus headache.

Before proceeding with a clinical description, it is necessary to call attention briefly to some of the anatomic peculiarities of this area and to the more recent discoveries in the field of general physiology which have a bearing on it.

Anatomic considerations will be limited to the course of a lateral nasal nerve in its bony canal, as found in a specimen used for serial sections. The accompanying illustration (Fig. 2) shows a transverse section through the external nasal branch of the lateral nasal nerve in its course through the bone. A communication of the canal with the nasal mucoperiosteum is also shown, through which a large branch is given off to the mucoperiosteum of the lateral nasal wall. It is believed that this orifice illustrates the area of the "tender spot"

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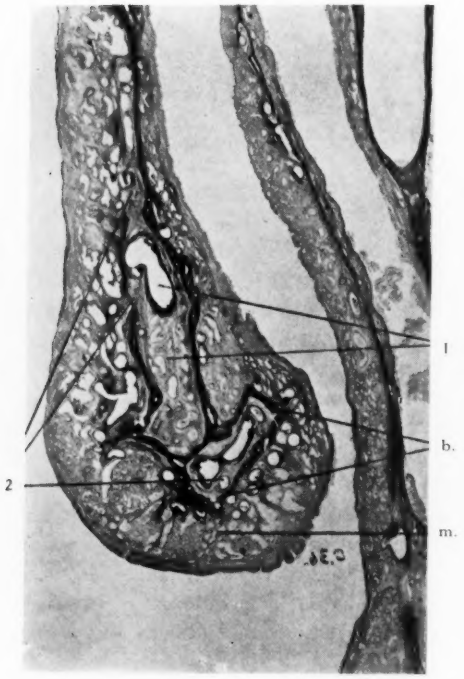

Fig. 1. (Mag. 10x). Cross section of middle turbinate showing the turbinate canal and vessels. 1. Middle turbinate artery and plexus about it. 2. Various smaller canals which contain veins and these in turn are continuous with those in the middle turbinate canal. b. = bone; m. = muco-periosteum.

which is often found clinically in these headache cases. Fig. 1 shows a cross section through the anterior tip of the middle turbinate with numerous bony canals, and therefore potential areas of pressure on nerves from congestion. Fig. 3 shows the lateral nasal wall of the block from which Figs. 1 and 2 were cut.

The name of Sir Thomas Lewis[4] is sufficient to direct attention to the outstanding work on peripheral vessel reactions in the human skin which he and his collaborators have been carrying on during the past decade and more. It is from him that we have received the valuable information that a histamine-like substance which he calls H-substance is formed in the tissues in the neighborhood of the minute blood vessels and causes their dilatation. A further study[5] of the tissue metabolites, as they are called, has led him to another interest-

ing discovery in connection with erythralgia. This condition is described as a flushing and a burning sensation in the skin of the legs on exertion. By the simplest of technic, but exceedingly clever deductions which one has come to expect from this observer, he shows that an irritating substance is produced in the tissues. It is in the nature of a tissue metabolite and in this case is responsible for the severe burning pain which is the most annoying symptom of this disease.

To sum up, it is evident that from stimulation by an external agent, substances may be formed in the tissues which not only influence the size of the blood vessels causing congestion, but that irritating substances may also be formed in the tissues which cause conscious irritation, i. e., stimulation of sensory nerves.

A considerable field of interest has therefore been opened up to the clinician, and if we can benefit our patients by reasonable postulates based on this work, we are surely progressing in the right direction. For example, we are justified in concluding that irritating substances are formed in the nasal mucosa from many causes; that they are neutralized or removed by means of a good circulation, and that local congestion may be due to local conditions and may not necessarily be dependent on any major disturbance of body function. Of all irritants which commonly influence the nasal mucosa, one of the most vicious is the toxin of the common cold, more especially that of the influenzal variety, which is particularly irritating to the peripheral nerves.

Within the past year and with the co-operation of the Department of Physiologic Hygiene and a grant from the Banting Research Foundation, some experiments were carried out with drafts. It was found that the draft from an electric fan directed on the legs increased the sensitiveness of the nasal mucosa to palpation with a probe.

All these results of physiologic experiment indicate a means by which the nasal mucosa may be rendered more sensitive and may result in excessive stimulation of the peripheral nerves in the nasal mucosa. An hypothesis is therefore evident by which headaches of nasal origin may be produced and a logical treatment indicated. The latter should be directed at improvement of the local circulation with clearing of irritating substances from the tissues and relief of congestion.

This headache of nasal origin is associated with changes in atmosphere. The high humidity and extremes of heat or cold bear a

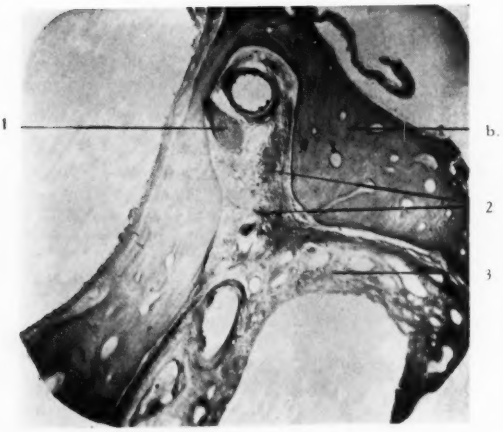

Fig. 2. (About Mag. 10x). Enlargement of the "tender spot" area, showing cross section of canal and foramen through which a lateral nasal branch is given off from the nerve to the lateral nasal wall. 1. Canal containing external nasal nerve and vessels. 2. Mucoperiosteum and vessels of nasal cavity. 3. Branch of nerve entering the nasal cavity. b = bone.

direct influence in producing it. Drafts, winds, stagnant air and cold feet are also very definite factors. Fatigue and excitement play a part and finally the common cold. The last requires special reference, being associated, as a rule, with the onset of cases of long or short duration, when a definite history can be obtained. The original exciting cause of the headache has been due to one bad attack of influenza or arose at a time when the individual suffered from a series of head colds. It may be twenty-five or thirty years since he first became aware of the headache, and it has recurred with greater frequency as time has passed. Another bad cold, perhaps, two or three years ago, has aggravated the headaches and it may be at this stage that the patient is occasionally incapacitated and may be forced to stop work for several days at a time. The increase in the headache is related to a fresh cold, as a rule, or to a time when the individual has been exposed to an epidemic of colds.

During all this time the recurrences of headache are precipitated by such slight causes that many patients may be unable to recognize the sources. Usually, however, it will be associated with a draft, a stuffy atmosphere, cold feet, a chill, a slight cold during an epidemic such as frequently occurs in the winter time. Changes of climate may be the factor. Truly, from such slight causes is this headache

precipitated at this stage that the unfortunate individual is the victim of headache most of the time. At night a change of atmosphere may be enough to wake him up with headache. It is no wonder that this headache is of such frequent occurrence in the community. It may be associated as a clinical entity with graver conditions, such as high blood pressure, etc., and yet be, comparatively speaking, unrelated to them. In fact, from observation extending over some five or six years, it is believed that few headaches involving the ophthalmic division of the fifth, with the exception of that relieved by correction of eye strain, can be eliminated from this category of nasal headache without the test described below. In the case of eye strain, if this type of headache is persistent after suitable correction, the test should be made for nasal headache as a probability.

Similarly, with occipital pain or headache, attention should be directed to this area in the nose as a possible cause. Occipital pain is often associated with arthritis and many other conditions, but the importance of a possible nasal cause should be definitely eliminated. The patient does not, as a rule, associate the various conditions noted above as the exciting causes of the headache, and it is not until treatment is under way that a clear history, such as has been mentioned, is given by him. It is therefore essential to eliminate the nasal cavity as a possible source of a headache involving the areas supplied by the trigeminus nerve before stating that the cause is not of nasal origin. An otherwise normal person may be suffering with headache involving one or more of the following—frontal, temporal, parietal, vertical or occipital areas of the head, or an aching pain behind the eyes. It has not been an uncommon remark of these patients to say "Why was I not told of this before? The doctor said it was probably due to so and so, but I got no relief." Not infrequently the nose has been examined thoroughly, but has not been considered the seat of the trouble, as no local lesion had been found and the sinus examination had been negative.

SYMPTOMS

The typical headache begins during a cold in the head. A feeling of fullness or tightness is felt between the eyes at the junction of the base of the nose and the frontal area. It extends over the frontal, temporal, parietal and vertical areas following the distribution of the ophthalmic division of the trigeminus. There may be an aching pain behind the eyes. It may also be felt in the occipital region. In some cases the scalp is exceedingly tender over the parietal and temporal areas. The headache is more frequently confined to one side, but with increasing severity may involve both. It may be per-

Fig. 3. Shows lateral nasal wall from which sections were cut for Figures 1 and 2. 1. Approximate area shown in Fig. 1. 2. Approximate area of lateral nasal nerve shown in Fig. 2.

sistent, but, as a rule, is intermittent and may develop in the daytime or at night. During the headache the patient feels a lack of energy and tired, but on lying down does not get relief. The usual remedies of aspirin, hot or cold applications to the fronto-nasal area, are of temporary value but in the more severe cases fail to bring relief. The headache may be of any degree of severity and may involve any one of the above areas separately from the others. Pain may be felt in the shoulder or arm on the affected side.

It may cease to bother the patient for a time, but, as a rule, he notices an increased frequency of the trouble with varying intensity. It may be aggravated by use of the eyes, owing to the close association of the sensory nerve distribution to the eye, but if an error of refraction exist and the correction fails to relieve the headache, this nasal source is the most likely cause. These frequently occurring attacks of discomfort may extend over a period of years. The patient feels energetic and normal as soon as the headache passes off, and this is true of the most stubborn and severe headaches from this cause.

Head colds always aggravate it temporarily, and not infrequently the headache may become more frequent and severe following these attacks. It may finally reach such a degree of discom-

fort that the patient is seldom free of it and has to give up work for several days at a time.

The various factors of drafts, cold feet, etc., already referred to, tend to precipitate the headache, and it is no wonder that the poor sufferer becomes discouraged and is finally suffering from headache most of the time.

The above history associated with the headache is frequently denied or has been forgotten by the patient when first examined and the diagnosis must rest on the simple test given below. Subsequently, as the headache begins to respond to treatment, the patient will give the history which has been indicated, but at first he often has been unaware of any connection between the headache and the various exciting causes. This must be borne in mind, as otherwise these headaches of nasal origin will be repeatedly missed. The diagnosis must depend on the test, repeated at intervals if necessary, and the course between treatments carefully noted.

In the typical case no discharge from the nose may be present unless it be a watery one during an attack; nor is there any evidence of sinus involvement, although if such be present it must be taken into consideration as an aggravating cause. There may be tenderness over Ewing's area beneath the floor of the frontal sinus or even over the anterior wall. In the past it has been the usual custom to surmise that some form of frontal sinus involvement is present, although there may be no evidence that such is the case. We are all familiar with a diagnosis of vacuum frontal headache, although there is no convincing evidence that such a condition can exist, more especially when no definite clinical signs of sinus involvement are found.

A cotton applicator introduced into the nasal cavity will indicate, as a rule, a hypersensitive condition of the mucosa. If the cotton be moistened with ephedrine and applied to the middle turbinate quite spectacular relief may be experienced by the patient in a few minutes. He will say in a surprised tone that "the headache is gone." For some time it was felt that this anterior tip of the middle turbinate was the only seat of the trouble, honeycombed as it is with minute bony canals for the passage of blood vessels and nerves and supplied largely by the anterior ethmoid nerve, a branch of the nasociliary, which in turn is a branch of the ophthalmic division of the trigeminal. However, as more of these headaches came under observation and results from treatment of this area were not entirely satisfactory, a further search was made for tender spots. This search was rewarded with the finding of an excessively tender spot just beneath the bridge

of the nose. On passing a cotton applicator soaked in ephedrin along the bridge of the nose in the narrow channel formed at the junction of the septum and the lateral nasal wall, the patient experienced a sudden severe pain, described as if a red hot needle had been suddenly pushed into his nose at this spot. In a few minutes, as a rule, great relief is experienced from the headache and the "spot" becomes less tender. This "tender spot" is situated over the course of the lateral nasal nerve, a branch of the anterior ethmoid, and in some cadavers examined it lies in a bony canal somewhat similar to the anatomic illustrations above. Presumably this nerve, or a branch of it, leaves the bony canal at this point in these cases and is subject to pressure from the vascular coat in the periosteum about it. The ephedrine causes contraction of the overlying veins and presumably those in the neighboring canal, and in this manner the pressure on the nerve is relieved, with relief of the referred pain or headache.[8] The result is quite spectacular and of diagnostic significance.

Little need be said on the subject of diagnosis. If the headache is relieved by the simple means described, the source of the headache has probably been found. In my opinion, no headache should be considered as due to some constitutional ailment or classed as migraine until this nasal examination has been carefully carried out and the effect on the headache or pain observed over a sufficient period.

The usual routine rhinologic examination is carried out, giving due weight to a possible sinusitis as an aggravating cause.

A cotton applicator is moistened in a 3 per cent solution of ephedrine in saline, and direct application is made to the nasal mucosa, noting any hypersensitive areas which may be present. These are found over the various bony canal openings which have been described in a previous paper[9] dealing with the "Blood Vessels of the Lateral Nasal Wall." Not infrequently an aggravation of the referred pain may occur as one or other of these "tender spots" is touched.

Particular attention must be paid to the inner surface of the bridge of the nose. It is here that the lateral nasal nerve, a terminal branch of the anterior ethmoid, is found. This anterior ethmoid is a terminal branch of the nasociliary and the close association with the pain behind the eyes is therefore evident. After the mucosa has shrunken following the first application of ephedrine, the cotton applicator is passed close under the bridge of the nose as high as it will safely go. Not infrequently the headache will begin to disappear in a few minutes.

The treatments should be carried on at one, two, three or four day intervals until the patient has passed through the various aggravating conditions mentioned above, such as changes in the weather, etc., and has remained free of the headache or pain. A very small percentage of the headaches treated have been obstinate.

In cases where headache has subsided under treatment, the patient should be cautioned to return for further treatments if the pain or headache returns.

SUMMARY

1. The source of a particular headache or pain of nasal cavity origin is described, involving the trigeminus nerve and also the treatment of this condition.

2. An hypothesis is advanced for the explanation of this headache or pain.

a) The irritation within the nasal mucosa from the products of bacterial invasion produces a hypersensitive or susceptible state.

b) As a result, substances normally produced in the tissues from external stimuli, such as changes of atmosphere, drafts, etc., cause excessive stimulation of the neurovascular mechanism and sensory nerves. The headache or pain which results is in the nature of a "referred pain" and involves one or more branches of the trigeminus.

c) The "tender spot" beneath the nasal bridge is believed to be due to pressure from congestion on the trunk of a hypersensitive nerve (a branch of the anterior ethmoid) in a bony foramen or canal, and this in turn may aggravate or produce the headache or pain.

3. The hypothesis applies to the pain associated with sinusitis and is believed to be more comprehensive than the generally accepted theory which is held at present. The latter is based chiefly on some form of obstruction to sinus drainage but fails to explain satisfactorily many headaches and pain associated with sinusitis.

In conclusion, I should like to express my appreciation and thanks to Dr. Duncan Graham for his cooperation and to Dr. H. H. Hyland and those in the Neurologic Division of the Department of Medicine who have been kind enough to cooperate in this work. My thanks are also due to Dr. P. G. Goldsmith and Dr. K. G. McKenzie for their interest and encouragement.

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VI

SURGICAL REMOVAL OF THE SPHENOPALATINE GANGLION*

Report of Three Operations; Elaborating an Original Technic to Expose the Pterygopalatine Fossa, Command the Internal Maxillary Artery and Its Terminals and the Infraorbital Nerve and Its Branches

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The importance of a trans-antral surgical approach to the sphenopalatine fossa was suggested to me by the fact that

1. Stubborn cases of fifth nerve type neuralgia in which the gasserian ganglion has been severed have been relieved by anesthesia or destruction of the sphenopalatine ganglion.

2. Infection in the sphenopalatine fossa, usually the result of injections, is not uncommon and has been fatal in spite of surgical measures heretofore employed.

3. The whole extracranial part of the infraorbital nerve is available.

4. The internal maxillary artery and most of its branches can be isolated and ligated.

5. The various branches of both the infraorbital nerve and the sphenopalatine ganglion are at hand for surgical disposition.

6. The sphenoid sinus can be entered and operated upon.

7. There are many other vital structures in the neighborhood that skillful surgery may approach.

8. The orbit is surgically available from below, even to the apex. This feature has been taken advantage of through a different incision in my recently published technic for the relief of pernicious exophthalmos.

In 1926[1] I published an operative approach by way of the maxillary sinus to the pterygopalatine and infratemporal fossæ. It

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was subsequently learned that Carnochan,[2] in 1858, had followed the infraorbital nerve from its exit at the infraorbital foramen to the foramen rotundum. His approach differed in that an incision was made through the cheek and the nerve was followed with sacrifice of the orbital floor.

In 1929 Vogel[3], following precisely by technic in the manner of approach, elaborated the details of dissection in the fossa and removed the sphenopalatine ganglia in nine living patients. He was interested in determining the condition of the ganglion in ozena and came to the following conclusions: 1. In genuine atrophic rhinitis there is isolated atrophy of the sphenopalatine ganglion. 2. Its disease is secondary to the ozena. 3. Because of the insignificance of the disturbance caused by its removal, he believes that its functions are quickly taken over by other nerves (perivascular branches).

Gussenbauer, Friedlander, Kocher, Lucke, Lossen, Braun, Krause and Lever all approached the pterygopalatine fossa from the side by a preliminary resection of either the temporal arch or the entire malar bone.[4]

In 1931, Braeneckner[4] described an approach from the side, through the skin, that included removal of the wall of the antrum.

In 1935, Auerbukh et al.[5] divided prior operations into three groups according to the approach: Orbital, transantral, zygomatic. They describe an ingenious method which has been worked out on the cadaver. It gains access to the pterygopalatine fossa from below by resecting the hard palate.

A number of fatal infections in the pterygopalatine fossa have occurred following injection of anesthetics or other fluids. These accidents have followed injections by way of the skin in the region of the zygoma and also those made through the mouth via the postpalatine canal or around the alveolar process.

The approach through the antrum, if employed early in such serious infections, should offer adequate surgical control.[6]

The technic of the operation, as first described by me in 1926, is given below with subsequent modifications.

The work is done under sodium amytal and scopolamin and morphin sedation and local anesthesia.

The Caldwell-Luc technic is used in opening widely through the canine fossa into the antrum, and from the antrum under the inferior turbinate into the nose.

The mucosal lining of the posterior wall of the antrum is circumbscribed by an incision down to the bone and is removed. The rest of the mucosa is not disturbed unless disease indicates the necessity for its removal. The posterior bony wall of the antrum is next removed and the periosteum enclosing the contents of the pterygopalatine fossa exposed. A strong solution of cocain is applied to this periosteum. Injection of the space for anesthesia may damage an artery and cause delay.

The periosteum is incised carefully and the underlying parts dissected with blunt instruments.

The internal maxillary artery is seen pulsating in a bulging arc across the field. It is followed to the nose without injury.

The infraorbital nerve is discovered lying across the depths of the upper part of the operative field. It is followed toward the foramen rotundum, which can be clearly seen.

Extending downward and medially from the infraorbital nerve near its exit from the foramen rotundum, the sphenopalatine ganglion is found. It lies just outside the nose, anterior to the base of the pterygoid process and almost directly posterior to the internal maxillary artery as it approaches the sphenopalatine foramen. (Fig. 1.)

The ganglion is freed by careful dissection with small hooks and forceps and is removed after its various branches have been severed. The artery or any of its branches may be ligated if required.

The infraorbital nerve may be severed at the foramen rotundum if advisable. The wound is closed as usual.

I am herewith reporting two cases operated upon in this manner. One of them had both ganglia removed at successive operations, more than three years apart.

REPORT OF CASES

CASE 1.—Male, 30 years of age. First consulted me October 30, 1930. An agreeable young man, with strong personality evident under a modest demeanor. Complaint: Severe pain in upper jaws and teeth, over, about and in both eyes, roughness of eyelids during an atttack when pain also radiates down the arms and extends even into the legs. Nauseated when pain becomes bad. Allergic history and diagnostic testing negative.

Has had a watery discharge from the nose that is more or less constant. Has about one cold a year. Ordinarily breathes freely through the nose, which swells and becomes stuffy prior to an attack of pain. Radiographs have shown no evidence of sinusitis. Tonsils have been removed and hearing is good. There is some tinnitus during attacks. No arthritis and patient is otherwise well.

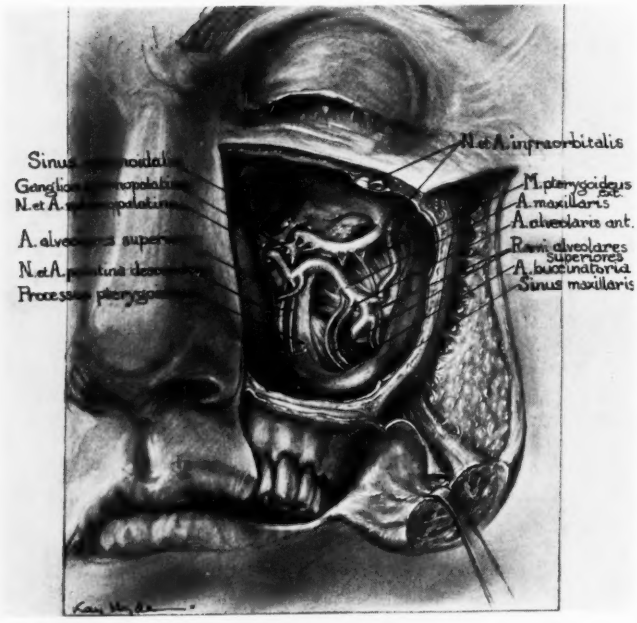

Fig. 1. The wide exposure shown here is, of course, from a laboratory dissection. It, or some modification, may be employed when malignancies or destructive processes minimize the importance of postoperative disfigurement. However, the approach described in the text is according to the Caldwell-Luc technic.

Examination disclosed nothing of importance except that cytologic study of the nasal discharge disclosed numerous polymorphonuclears on the right side. Test of the right antrum showed macroscopically sticky mucus and microscopically a few mononuclear and polymorphonuclear leucocytes. The left antrum showed only a few mononuclears. Operative procedures to clear up the low grade sinus infection were of no avail in relieving the distressing and incapacitating pain.

His condition finally became so pitiful and the employment of drugs so alarming that it was decided to remove the sphenopalatine ganglion on the left or worse side. This was done January 12, 1931, according to my technic which has been described. He left the hospital without pertinent incident on January 30, 1931.

His letter of February 9th says: "The pain in the left cheek, nose, eye and upper jaw is slowly going away, but is still constantly discomforting and at times unbearable. Every few moments there is some sort of disturbance in the left cheek or left upper lip as though a hot needle were passing through it, or sometimes like a bee buzzing in a flower. The needle effect is painful; the buzzing sensation is only uncomfortable. The left eye aches and is slow to adjust itself to the light in

the morning. The teeth under the incision ache most of the time, more after eating and drinking. The left roof of the mouth is numb, yet at times there is feeling of a hot needle running back and forth in it. The external swelling is still apparent but gradually reducing. I believe that as soon as the operative pain disappears there will be no further trouble."

In another note of March 18, 1931: "I am working eight hours a day. We are going out to dinner, playing cards, and so on. I am sorry the pain does not go away. The left cheek, lip and eye continue disturbed. The headache has never left. It is intense and constant. We have done everything we can, so I am going right ahead disregarding the pain as far as possible."

On April 30, 1931, he again writes: "The operation has proved to be a complete success. There is none of the old pain in the left side. The nausea has completely disappeared. There has been no more pain in the arms or fingers. Occasionally the old pain comes back full length in both legs. All medicines have been discontinued. Recovery from the operation has been very slow. I had the left antrum washed out. The water returned clear. Aside from the slow recovery I am delighted with the operation. The pain has stopped completely on the left side. If the right side continues painful would you recommend the removal of the right ganglion? It is fine to be at work again."

September 16, 1931, he says: "I am writing to tell you that the removal of the left ganglion has given complete relief from pain on left side and to ask your advice about the right side. Numbness on the roof of the mouth is noticeable only in eating; then pressure on the teeth under the incision is unpleasant.

"The right side is the same as it has been for the past ten yeaers, and I am often tempted to ask you to remove the ganglion on that side. The right eye burns and the lid feels rough and the surrounding area feels bruised. Intermittently there is pain in the right forehead, a headache and pain in the right jaw the same as toothache. The pain in the jaw skips around, sometimes upper, sometimes lower, usually front upper and back lower. In a period of pain of more than usual severity I am nauseated. Flare-ups of severe pain last a day or two. There is practically no freedom from pain. The use of the eyes seems to make the pain worse. Long auto or train rides invariably set it off. Reading, playing cards or tennis do likewise. The situation is entirely different than before the removal of the left ganglion. Then it was impossible, now it is annoying. On account of daily swimming and tennis, I am in splendid condition. I would appreciate very much your advice."

I did not hear again from the patient until 1934, when the following letter came:

June 12, 1934. "For the past several months the pain on the right side of my head has been very severe. The left side is entirely free from pain and there are no ill effects from removal of the ganglion. The right side, however, is very bad. The symptoms are the same as for years except that the pain has been more constant, more severe, there being practically no free periods. The pain is in both upper and lower teeth on the right side, the right eye and in the right forehead above and about the eye. The right eyelid burns and during an attack the eye waters. When the pain is severe I am nauseated, recently vomiting for about three days. Three weeks ago the doctor here cut a window in my right antrum. I am writing to know how you would feel about removing the right ganglion for me. If the pain continues to be as severe, I must do something to relieve it."

June 29, 1934. My answer: "Your letter has left me in a quandary as to how to advise you. I am happy that what I did for you has given you so much relief. I admit that I was loathe to do that work and am even more anxious to avoid working on the other side. I think that in as much as you have had an operation recently you had better give it time to see what the ultimate result will be."

July 16, 1934. Without having consulted me further at this time he had another surgeon remove his right sphenopalatine ganglion.

Following this operation there was no change in the character or severity of the pain in the right side of the head. Pain in the legs increased and pain in the hand returned. There has been no numbness about the head similar to what followed the operation on the left side. He was advised by a neurological surgeon to have something done to the right cervical sympathetics.

December 5, 1934. The patient consulted me again. He was in great distress with the right side of head and body as outlined. It had become necessary to take opiates constantly; he was taking morphin and codein up to 12 grains per day; also other opiates at intervals.

January 21, 1935. I operated upon the right sphenopalatine ganglion and he left the hospital in about ten days without unusual incident.

February 11, 1935. A letter from Carmel says: "We made the trip down as well as could be expected. While the eye, cheek incision and jaws are full of pain, it is easier to stand. You will be glad to know that I went twelve hours last night between hypos. We have this thing beaten. All that is necessary is to hang on for a little while."

March 4, 1935. "I have been home two weeks and suppose I am making fair progress. The right eye, cheek and lip are still sensitive to touch. I have not been to the office yet, though have worked in the garden some."

February 25, 1936, he writes: "I am happy to report to you that the work you did in January, 1935, has given complete relief from the old pain. I am now free from the old pain on both sides. I have since had no craving for narcotics nor have I had any in any form. I am working every day and have gained thirty pounds."

May, 1936, he consulted me. He is a healthy appearing, hard working, successful business man. At intervals he has pain and disturbance in the right cheek. It seems to have some relation to taking cold. Stuffiness of the nose precedes the attacks. We, therefore, cannot call him well. He is infinitely better, however. Except for some altered sensation to touch over the cheek and hard palate, there is no evidence of nerve disturbance.

Both sides of the nasal fossæ are equally sensitive to touch. The turbinates on both sides shrink immediately and normally where neosynephrin is applied.

The sympathetic control was apparently quickly re-established. This has been described by others and ascribed to the assumption of control of the sympathetic fibers following the blood vessels.

The following information was contained in a note received received from the patient in December, 1936: He is almost over his troubles, is working every day and quite a few evenings, including a lot of traveling on business.

CASE 2.—Mrs. B., age 55, entered Stanford Clinic July, 1931. Complained of pain in the left side of face which had persisted for four years. Pain began in left zygomatic region, gradually increasing in severity. After three months the left gasserian ganglion was operated upon. This relieved the stabbing feature but dull pain persisted. A sympathectomy was done two and one-half years ago and this was followed by injections without relief. The patient was practically helpless.

A nervous woman, complaining bitterly of pain. Showed no organic disturbance except for loss of all sensation over the region of distribution of the left fifth nerve. Diagnosed trigeminal neuralgia, with confusing psychic disturbances.

October 29, 1932. Dr. Reichert did a diagnostic block of the left cervicothoracic sympathetic chain. There was some relief. On May 22, 1933, this was followed by removal of the cervical sympathetic chain and carotid sinus.

August, 1935, left sphenopalatine ganglion was concainized, without improvement. The sphenopalatine ganglion was later injected with novocain by Dr. Rixford. There was definite temporary relief.

October 16, 1935, I removed the left sphenopalatine ganglion according to the technic described. There was some pain complained of when the ganglion was manipulated. She was discharged from the hospital without intervening incident, on October 20, 1935.

April 9, 1936. A letter from Dr. Rixford tells the following story: "Here are the results of my examination: Complete anesthesia to pain, temperature, pressure, etc., over the distribution of all the branches of the fifth nerve. Hearing is not impaired on the left side. The left pupil is active to light and accommodation. Tearing is about the same in both eyes. There is more or less complete anosmia on the left. Taste to salt, acid, sugar and quinine is markedly diminished on the left, both on the soft palate and the tongue. Gag reflex is absent on the left, present on the right. Pain and temperature sensations are absent on the left half of the tongue.

"Mrs. B. appears to me to be much improved. But she still complains of a constant, dull pain in the region of the left zygoma. This pain radiates to the left parietal region. She complains of parathesias, such as prickling sensation along the lower jaw. She states that at times this same sensation is present over the left eye and rarely over the rest of the distribution of the fifth nerve. Also, the left eye appears considerably deeper in the orbit than the right, and there is slight drooping of the upper lid. She still has a great deal of difficulty in swallowing anything but liquids, and she states even liquids cause severe pain. She is unable to chew any solid food so she confines herself to liquids and mushy solids. However, she has gained four pounds in weight in the last five weeks. She also complains of a severe photophobia in the left eye. This does not start the pain in the zygoma as it used to. The severe jumping pain that was the worst of all has been absent since our resection of the sphenopalatine ganglion. She is now able to sleep as much as six hours at a time if she takes two or three three-grain capsules of sodium amytal. Up to five weeks ago she used half a grain of morphin as well as two sodium amytals to get three hours' rest. She has had no morphin for five weeks.

The following communication has since been received from the attending physician:

"I have not seen Mrs. B. for quite some time. When I last saw her she was about as miserable as ever. She still complains of a constant pain over the distribu-

tion of the left fifth nerve. The pain is almost always present, but is worse at some times than at others. There seems to be no relation to temperature or time with the exacerbations. She takes large quantities of sodium amytal but for the last several weeks has not required opiates. She did require large doses of opiates for several months since her operation. She went without opiates for three months or more immediately following resection of the ganglion. The severe, jumping pain of which she complained before the resection of the ganglion has never returned. She has had two neurologists go over her since her last visit here and has had a course of x-ray therapy with far less resultant benefit than from our operation.

"I am now firmly convinced that her pain is of central origin, as every sensory or possibly sensory nerve on the left side has now been cut and she still has her pain. Incidentally, she has lost no weight and looks as well as she ever did since her first visit to my office."

STANFORD UNIVERSITY HOSPITAL.

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VII

BLOOD TRANSFUSIONS IN DISEASES OF EAR, NOSE AND THROAT*

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A scientifically administered tranfusion is the nearest approach to a perfect tonic available in modern medicine. Once used as a futile gesture in the final fight against a hopeless situation, today a blood transfusion is a valuable therapeutic measure in the pre-operative and post-operative management of promising cases.

The transferring of blood from the donor to the patient is more than a simple intravenous medication, and the potential dangers should modify our judgment in the selection of the time and frequency of its administration. If possible, transfusions should be given by surgeons who confine their efforts to this specialty or who, through experience, are apt in its administration. The indications for a transfusion in our specialty are:

Hemorrhage of any type, spontaneous, accidental or operative, that acutely reduces the hemoglobin to 75 per cent, the clinical symptom of importance being air hunger, due to the loss of oxygen carrying ability of the blood. Repeated minor hemorrhages over a protracted period or a continued destruction of blood by toxins or poisons to a poverty of 50 per cent hemoglobin demands a transfusion prior to surgery.

REPORT OF CASES

CASE 1.—J. B., age 4, was operated on for the removal of tonsils and adenoid at 8:00 A. M., May 6, 1935. The child reacted favorably from the operation. At 9:30 A. M. she vomited two ounces of dark bloody stomach contents and a similar quantity at 1 P. M. After a short nap at 4:30 P. M., the child awakened and vomited approximately sixteen ounces of stomach contents containing bright blood. Three minims of surgical pituitrin were given hypodermatically every fifteen minutes for three doses in an attempt to control the hemorrhage from the adenoid. Again at 6 P. M. the child emptied her stomach of seven ounces of fluid containing dark blood. The child became very restless and showed signs of air hunger. Examination of the throat revealed a large clot in the adenoid region with

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a show of blood in the lower half of the right tonsil fossa. Under general anesthesia, a postnasal tampon was inserted and a vein was ligated in the lower part of the right tonsil fossa. Blood count: Hemoglobin 70 per cent, R. B. C. 2,800,000. The child was of type four and a compatible donor was tested for a transfusion. The multiple syringe method of Lindemann was the procedure of choice and at 9:30 P. M. 120 cc. of whole blood were administered. The child showed immediate relief of the air hunger. After an hour's watching to determine the thoroughness of the control of the hemorrhage, she was allowed to go to sleep. Her night's rest consisted of short naps, but there was no further bleeding or symptoms indicating shock. The postnasal plug was removed fourteen hours after its insertion and the child made an uneventful recovery.

CASE 2.—J. C., age 5, referred for the removal of tonsils and adenoid on June 18, 1934, with the history of scarlet fever the past March, since which time she continued to have recurrent attacks of tonsillitis complicated by an acute purulent sinusitis.

Examination.—The nasal cavity contained a mucopurulent discharge and the tonsils were of the active glandular type. A roentgenologic study revealed a persistent infantile type of sinuses with a cloudiness (grade 3) of the antra. Blood count: Hemoglobin 60 per cent, and R. B. C. 3,200,000. The child was hospitalized and daily nasal treatments instituted with slight improvement of the character of the nasal discharge. On June 27, one week after admission, the child was given 150 cc. of whole blood by the multiple syringe method. The tonsils and adenoid were removed and the antra drained on June 29, the only complication being an excess of the sanguineous quality of the discharge from the sinuses.

Sepsis.—Irrespective of the blood picture, a transfusion is indicated whenever a septic patient is not doing well. That demand may require a daily transfusion or at intervals up to five days. Compatible red blood cells live and function as long as 93 days, but the serologic improvement is transient and of little value after the fifth day. The symptoms and signs of infection, such as chills, fever and depression, are better indicators than the white blood count with its differential or the red blood count with its hemoglobin content. The latter is usually low, as there is a secondary anemia in both the acute and chronic septic patient. Part of the improvement that follows a transfusion is the result of the improvement of the secondary anemia.

CASE 3.—R. M., age 7, referred June 24, 1935, for a mastoid operation with a history of an acute middle ear infection for the past three weeks. Temperature on admission was 104, pulse 116, blood count: Hemoglobin 82 per cent, R. B. C. 4,560,000, W. B. C. 13,150, small lymphocytes 16 per cent, neutrophiles 83 per cent (juveniles 5 per cent, stabs 15 per cent, segmented 63 per cent) and basophiles 1 per cent. The child was profoundly ill and looked the part. In spite of the average red blood count and hemoglobin a transfusion was indicated and given with beneficial results. The following day the mastoid was operated upon. The postoperative convalescence was stormy with a continuation of the septic temperature. On the third postoperative day the child complained of headache and vomited his supper. Blood count: Hemoglobin 92 per cent, R. B. C. 4,840,000, W. B. C. 16,050,

small lymphocytes 22 per cent, neutrophiles 78 per cent (juveniles 5 per cent, stabs 45 per cent, segmented 28 per cent).

Fourth postoperative day—Developed an abdominal pain, which required a consultation with the surgeon to eliminate an acute appendicitis. Blood count—Hemoglobin 87 per cent, R. B. C. 4,500,000, W. B. C. 12,000, small lymphocytes 27 per cent, neutrophiles 73 per cent (juveniles 3 per cent, stabs 13 per cent, segmented 57 per cent).

Fifth postoperative day—Pain over right eye more constant and severe. Blood count: Hemoglobin 86 per cent, R. B. C. 4,500,000 W. B. C. 9,800, small lymphocytes 23 per cent, neutrophiles 77 per cent (juveniles 3 per cent, stabs 8 per cent, segmented 66 per cent).

Sixth postoperative day—Surgeon satisfied that abdominal pain was not from a surgical appendicitis. Blood count: Hemoglobin 80 per cent, R. B. C. 4,160,000, W. B. C. 16,400, Polynuclear 71 per cent, small lymphocytes 28 per cent, eosinophiles 1 per cent.

Seventh postoperative day—Nauseated, but did not vomit. Less pain in head. Wound still draining profusely.

Ninth postoperative day—Up in wheel chair. Temperature 99°. Wound only discharging through rubber tube drain.

Twelfth postoperative day—Second transfusion of 300 cc. of whole blood. Wound still discharging more than average. A mild headache at times during the day. Discharged from the hospital.

This child presented a true Gradenigo's syndrome, beginning with paralysis on the 21st day and lasting until the thirty-ninth postoperative day.

Comment.—A blood transfusion is indicated as frequently in a septic case as the surgeon considers his patient not doing well, irrespective of the blood count.

Diseases associated with a decrease in the number of blood platelets (idiopathic purpura hemorrhagica and symptomatic purpura hemorrhagica) produce bleeding either spontaneously or surgically. Occasionally an unfortunate operation is performed for what appears to be a chronic tonsillitis and a delayed study to ascertain the cause of the hemorrhage results in the finding of this blood dyscrasia. The life cycle of platelets is less than five days, thereby requiring frequent transfusions for this blood disaster. Occasionally preliminary venesection is demanded before subsequent transfusion to prevent plethora.

Hemophilia may be either true or transient. Large transfusions are necessary to supply sufficient platelets or the humoral elements (fibrinogen, calcium or prothrombin) when surgery is demanded or uncontrollable hemorrhages have followed accidents or surgery.

Agranulocytosis.—Repeated transfusions have been employed as a means of supplying leucocytes until hemopoietic stimulants have

had an opportunity to increase the number of white cells. In contrast, transfusions are of little value in supplying leucocytes during acute infections.

METHODS

The blood may be either transferred in a natural state or modified by the addition of sodium citrate.

Direct anastomosis of the vein of the donor to the recipient is a very technical operation and difficult to control. No method is available of measuring the volume of blood transmitted and the only practical test is a clinical observation of the improvement of the patient.

Modified Blood—By the addition of citrate to a known quantity of freshly drawn blood, a fluid easily handled and capable of transportation is obtained. The introduction into the recipient is simple and can be stopped at the onset of any undesirable symptom. Many surgeons prefer this method, claiming for it greater ability to increase coagulation, whereas others condemn it as an unscientific soup, devoid of those antigens so valuable in unmodified blood. There are more reactions following the use of citrated than of whole blood. Some surgeons welcome a slight reaction in both the patient with a hemorrhage and a septic case.

Unmodified or Whole Blood.—Many varieties of apparatus have been presented with claims of their simplicity, but as yet the multiple syringe method of Lindemann remains the safest and most expedient method.

TYPING

To Jansky and Moss belongs the credit of classifying human blood into groups, but we must depend upon the interpretation of the well trained laboratory technician to see that the blood of our donor shall blend with that of our patient. There are four groups of human blood and in order to simplify their designation the National Research Council renamed the groups with letters to avoid the confusion produced by the different numbers used in the two older classifications.

Official	Jansky	Moss
O	I	IV
A	II	II
B	III	III
AB	IV	I

A safe transfusion is dependent upon the presence in the blood of agglutinins and agglutinogens, the former distributed in the serum and the latter contained in the blood cells. "These substances are not all present in the blood of each individual, but occur in such a manner that it is possible to place almost every human being into one of four groups, according to the presence or absence of the specific agglutinins or agglutinogens. For purposes of orderly thinking, the agglutinogens may be conceived of as of more prominence than the agglutinins and are usually designated by capital letters."

SELECTION OF DONOR

Group Equivalent			Group Content and Interaction	
Jansky	Moss	Landsteiner	Serum	Cells
I	IV	0	Contains agglutinins A and B. Agglutinates cells of other three groups.	Contains no agglutinogen. Not agglutinated by any serum.
II	II	A	Contains agglutinin B. Agglutinates cells of Jansky III and IV. Moss I and III. Landsteiner B and AB.	Contains agglutinogen A. Agglutinated by sera: Jansky I and III. Moss III and IV. Landsteiner 0 and B.
III	III	B	Contains agglutinin A. Agglutinates cells of Jansky II and IV. Moss I and II. Landsteiner A and AB.	Contains agglutinogen B. Agglutinated by sera: Jansky I and II. Moss II and IV. Landsteiner 0 and A.
IV	I	AB	Contains no agglutinin. Agglutinates cells of no group.	Contains agglutinogens A and B. Agglutinated by sera of other three groups.

A great responsibility should be appreciated by the laboratory technician and the surgeon. The blood of the donor must blend with that of the patient, for incompatible blood may result in death. The donor should be free of syphilis, malaria, asthma or any allergic sensitivity and be rich in hemoglobin. In certain instances, it is helpful if the donor's blood contains an abundance of immune bodies. The donor should be young, for the blood of the aged is poorer in antigens. It is not only necessary that the donor and the recipient be of the same group, but their blood should be cross matched and the time consumed in this matching is well spent. When the same donor is used for subsequent transfusions, this vital procedure should precede each giving to the patient. The donor never changes his group, but circumstances, especially infection, may produce incompatibility.

Reactions may be manifested by a chill, rigor, fever, nausea, dyspnea, cough or headache and may terminate in death. One hundred cc. of poorly matched blood may produce a reaction with a hemolysis from which the patient could recover, whereas an unfortunate administration of 500 cc. would produce death. Jaundice and hematuria are evidence of hemolysis, and embolism indicates massive agglutination.

Stetson divides the reactions into (1) hemolytic, (2) proteolytic. The former are the result of poor matching or from using unmatched blood, or because the recipient is suffering from a disease in which an active hemolytic agent is breaking down the cells.

Proteolytic class embraces three distinct types:

Febrile (the protein content of the donor's blood influences the type of reaction).

Protein (the dermal reaction of true allergic phenomena with or without systemic reaction).

Anaphylactoid (some of the reactions are of the true protein type and possibly some due to minor agglutinins).

CONCLUSIONS

Transfusion of human blood, a life saving measure, is one of the advances of modern surgery, giving to the patient on demand that which he cannot instantly supply himself.

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VIII

EIGHTH NERVE HIGH TONE DEAFNESS FROM A NUTRITIONAL STANDPOINT

GRANT SELFRIDGE, M.D.

SAN FRANCISCO

This article is written for internists as well as otologists with a plea for early recognition of this condition when loss of high tones between cycles 8192 and 16384 is first shown, for it is in the early stages that treatment promises most. A carefully taken radiogram on an audiometer with a range to 16384 cycles may be as valuable to the clinician as many another mechanical aid to diagnosis.

Degeneration of the eighth nerve begins about the same period of life that presbyopia, cardiovascular and other degenerative conditions are found.

As Crowe says, high tone loss is present usually in the fourth decade of life, and becomes progressively worse during the succeeding decades. I have, however, one patient under treatment, aged twenty years, who shows a beginning loss of high tones, associated with Meniérè's symptom complex, that appeared at the age of nineteen (to be reported in full later in this paper).

Nothing has as yet appeared in medical literature of value in treatment, except in some cases where acute infections seem to play a part, and even here the infection probably plays a röle secondary to the nutritional deficiency. Crowe[2] says, "as regards the röle of vitamins and dietary insufficiencies and the use of salicylates and other drugs in the etiology of deafness, it is possible to learn much by feeding animals with special diets."

While toxic neuritis from focal infections, described by Duel, usually clears up promptly, some infections, as influenza and mumps, not only appear to play a definite part in nerve degeneration but the defect appears to be permanent.

As to the effect of chronic infections, McMurray in a recent article reports cases having Meniérè's complex, associated with high tone loss. The clearing up of their infections improved the Meniérè's symptom, but he says in a personal communication, "no improve-

ment was noted in the hearing after the removal of foci of infection. It has been my experience that while the vestibular function has been restored the cochlear has not improved."

Albert Gray[8] speaks of otosclerosis as a nutritional disease and infers the same in his description of the degenerative changes observed in the nerve studies of one of his cases. He thinks the changes are due to vasomotor variations preventing the proper nourishment of the structure involved. He appears to be unwilling to accept the idea that "deficiencies of the internal secretions of the various glands" play any part in the subject. He says, "the subjects of otosclerosis are frequently and indeed usually in perfectly good health, a condition hardly likely to be present if the internal secretions were deficient or excessive in quantity." A somewhat amazing statement in view of the masterly articles of Drury[5] on the relation of the ductless glands to deafness!

I shall report later several cases with basal rates from —15 percent to —22 percent that were not benefited by thyroid, and in whom no apparent change in their audiograms followed thyroid medication, but in which correction of the deficiency resulted in marked improvement.

The nutritional studies of Nina Simonds, the blood chemistry of Becks,* the sugar tolerance studies, basal metabolic rate, and allergic tests carried on in a series of cases of my own, show pretty definitely the fallacy of such statements as made by Gray.

A careful study of Drury's articles gives no real clue to the action of the different hormones in the various metabolic processes, but the evidence in other literature seems to indicate a close relationship between the hormones and vitamins, and as Zondek[22] (Page 92) says, "both being catalysts acting upon the cell surface are in my opinion maintained there in a state of equilibrium. Thus, if the vitamin supply is defective, secondary changes occur in the production or secretion of the hormones." Various authors, notably Funk, Douglass, R. R. Williams, Crowell,[6] and McCarrison,[15] have observed changes in the thymus, adrenal and sex glands, all indicating the need of vitamins, especially the factors in the B complex.

Lepkovsky and Evans [13] reported changes in the blood sugar curves in B deficiency and recently Williams and his coworkers pointed out the influence of large doses of B_1 crystals in lowering the high sugar curve in cases of diabetes.

*Personal reports of a series of cases studied from a nutritional standpoint by Dr. Simonds, and blood chemistry studies of these cases by Dr. Becks.

There appears also further evidence on the influence of vitamins in other metabolic processes, i. e., especially in lowered basal rates described by Hayden[11] and Kroll, where, in cases of pernicious anemia the basal rate was as low as 35 percent minus and where the clinical appearance suggested marked hypothyroidism which changed to almost normal on the proper treatment of the pernicious anemia.

Quite recently (Oct. 9, 1935) I read a paper before the staff of the Southern Pacific General Hospital (San Francisco) in which I attempted to draw a parallel, from some of the literature on general diseases now being classified among partial or complete deficiency diseases, and certain conditions of the ear, nose and throat and particularly of the eighth nerve.

In the conclusions of that article the following points were submitted:

First, that the literature quoted and unquoted furnished sufficient evidence that many diseases of man have defective nutrition as a background.

Second, that there is slowly accumulating evidence that lowered nutrition plays a definite part in certain diseases of the ear, nose and throat that come within the classification of partial deficiency states.

Third, that affections of the perceptive organ of hearing, aside from toxic neuritis, have a definite relation to deficiency states.

Fourth, that attention to an optimum nutrition can delay the progress of eighth nerve degeneration, if faults of nutrition are recognized in early life, and permanently corrected.

Fifth, that it is possible after the fifth decade of life, to ameliorate the condition of an individual's hearing, provided the areas of voice perception, i. e., between 240 d.v. and 4096 d.v. are not hopelessly damaged.

This concept of metabolic disturbances in relation to the ear, nose and throat was formulated by me fully twenty years ago, and has been substantiated by the careful nutritional study by Nina Simonds, of a series of my cases of sinus diseases, allergy and deafness.

She studied the dietary history as far as obtainable from infancy to the age of twenty, and from twenty onward, taking up, first the carbohydrates, fat and protein intake; second, the amount of calcium, phosphorus, copper and iron in the food; third, the amounts of vitamins A, B, C, D, and G, and fourth, whether the diets were acid ash or alkaline ash.

In the chronic progressive deafness cases, especially otosclerosis, as well as in cases showing loss of high tones, she found that from infancy to twenty years, the carbohydrates were high, fats low, proteins normal. From twenty years to the present age, carbohydrates were fair, fats fair, proteins low to fair; in the majority of cases the calcium was low, phosphorus fair, copper and iron normal.

The vitamins A, B, C, D and G in both age groups were as follows:

A, in 50% was low, others fair; B, all low; C, all low; D, all low; G, 50% fair; the rest normal. As to acid ash and alkaline ash, half the cases were acid, half were alkaline.

Pretty much the same picture is found in the nutritional study of nerve deafness cases as appears in the otosclerotics, i. e., in the infancy to twenty year group, the carbohydrate-fat intake may find the former low to high, but with the fats low and proteins in sufficient quantity.

It appears quite evident in the survey already done by Simonds of otosclerotics and nerve deafness cases that their nutrition is very decidedly suboptimum. It is true that in a nutritional survey it is a very difficult matter to uncover all the facts, even when carried out by an expert, and unless one is able to have on his staff an expert dietitian, one can only arrive at an approximation of the deficiencies present.

In recent months accurate laboratory methods promise to supplant haphazard guesses in the treatment of deficiency diseases.

I refer to the use of the Birsch-Hirschfelt visual photometer in the determination of vitamin A, which measures the regeneration of the visual purple, described recently by Jeans and others, and the biophotometer recently on the market, an instrument perhaps more readily used and perhaps more accurate than the Birsch-Hirschfelt instrument.

For estimation of vitamin B a urine test has recently been published by Harris of Cambridge. Helmer and Roscoe have both presented methods for the urine check and still more recently Peters of Oxford has called attention to Johnson's method of determining B deficiency in the blood through the isolation of pyruvic acid. For determining vitamin C a urine test has been devised by Harris and Ray and a blood test originated by Farmer and Abt. For vitamin D there is the study of the blood phosphotases mentioned also by Professor Harris.

Much should be learned from the use of these methods, especially in cases of multiple sclerosis and in cases of marked bradycardia, as well as in obscure cases of eighth nerve involvement.

My energies were decidedly activated in January (1935) when Profant brought to my attention the remarkable results he had obtained in a case of unilateral nerve deafness associated with Ménière's disease, following the plan recommended by Furstenberg, i. e., dehydration from the use of salt-free diets and large doses of ammonium chloride.

I did not see then nor since, how sodium binding or water binding could play any part in the demyelinization of either branch of the eighth nerve, especially the acusticus branch, and even though I treated some of my early cases, with some improvement following Furstenberg's plan, I abandoned it, first because most of them complained bitterly of the stomach upsets from such large doses of ammonium chloride, and second, because I felt there are perhaps other and more important factors involved in the degenerative process; and that Furstenberg's findings represent a separate and distinct picture.

It became quite evident to me, after hearing Professor Drummond's lectures in San Francisco in 1934, that vitamin B deficiency might be at least one of the complex factors in the production of eighth nerve deafness. This was accentuated by Peters and Thompson's work, indicating that in B deficiency there may be a local accumulation of lactic acid in various parts of the central nervous system. This explains the opisthotonus observed in B_1 deficient pigeons. One can describe this as a local acidosis caused by lactic acid. Quite recently Peters[18] has elaborated this idea in an article on "The Biochemical Lesion in Vitamin B_1 Deficiency" and from the work submitted seems to "present conclusive proof that lactic acid does accumulate in certain areas of the brain and that vitamin B_1 was concerned with both oxidation and carbohydrate metabolism; the essential part it played was as a catalyst in the oxidation of pyruvic acid."

Quite recently Sherman and Elvehjen[21], in an article "In Vitro Studies on Lactic Acid Metabolism in Tissues from Polyneuritic Chicks," confirm Peter's work.

From the above facts, coupled with the report of Colonel McCarrison that normal cell growth of tissue does not occur in the serum of chickens on a B_1 deficient diet, and T. Ono's[17] studies on the eighth nerve in vitamins B_1 and C deficiency, showing degeneration of the nerve in such deficiencies, I formulated the following theory: That

in the majority of cases of degeneration of the eighth nerve, especially with loss of high tones, the degeneration was due to the accumulation of lactic acid in the sheath and the myelinated substance of the nerve, and that the lactic acid coming in contact with the highly complex protein fats composing the myelin substance probably brought about the degenerative changes in the nerve.

In the early part of 1935 I decided to test out the theory on a series of clinical cases in the different decades, from the second to eighth, and being somewhat uncertain with what B fraction to begin, I consulted with Lepkovskky, who had worked for years on vitamins with Herbert M. Evans, Department of Biological Research, University of California. He said that two young chemists in Berkeley had been recovering the B complex from rice bran, and he "would advise using the entire complex, because if you used only the Lilly's B_1 absorbate you might miss the most important factor." This plan was followed, but in the past months we have also used an intramuscular preparation of B_1 solution furnished by Lilly and Company, and also Lilly's B_1 absorbate, in order to determine the efficacy of B_1, and to compare these results with those obtained with the entire complex. These cases will be reported later with their audiometric charts.

The findings were so encouraging that in June, 1935, under the supervision of R. Archambeault, a young chemist at the Harriman Research Laboratories, Southern Pacific General Hospital, we put a series of sixteen rats on B_1 deficiency, fourteen on B_2 deficiency and twenty rats served as controls. In 1935, several rats were turned over to Dr. W. P. Covell, working under a grant from the Coleman Fund at the Hooper Foundation for Medical Research, University of California, for perfusion and preparation for histological studies.

About April, 1936, I saw a few animals on the filtrate factor of the vitamin B complex of Lepkovsky and was so struck with the amount of edema of the auricles, and about the mouth and feet, I became convinced immediately that the B factor would be of prime importance in causing the demyelinization of the nerve.

Shortly after, Professor Herbert M. Evans gave us two animals with this type of deficiency which were sent to Doctor Covell for perfusion. Several other animals were furnished for study from Doctor Lepkovsky's laboratories, Department of Animal Husbandry, Poultry Section, University of California.

We have also received several chicks for study, from the Poultry Division, College of Agriculture, University of California, the report of which will be made by Doctor Covell at a later date.

It appears that one of the filtrate factors produces edema, dermatitis and many other conditions in rats, and another factor produces a similar condition in chicks. Doctor Covell's study of the B_1B_2 and B filtrate factors in these animals and chicks will also be published at a later date.

We have apparently been able to produce a progressive degeneration of the eighth nerve in animals beginning with B_1 deficiency. The changes are a trifle marked in the B_2 group, and more absolute in the filtrate factor deficiency group. A more dramatic picture could not be imagined than the appearance of these rats and chicks in the end stages of B filtrate factor deficiency, especially the chicks. They are flattened out, almost completely paralyzed, unable to move their heads, wings or feet; but when a small portion of the filtrate factor is put down their gullets, within two hours they are up on their feet picking up food, and in twenty-four hours have gained as much as twenty-seven grams in weight. The animals put on B_1B_2 deficiency diets came from the animal houses of Professor Evans' Department of Biological Research and are a strain equal to, if not better than any others in this country. Their dietary formula also came from the same laboratory. It is my impression that animals of good strain and carefully fed over a period of years are not apt to show the ear diseases described by Lurie. His animals came from here and there and he cannot prove how far from normal their diets may have been. It is true, of course, that some of our animals on deficiency diets showed acutely inflamed ears, but these were discarded and only the normal ears were studied.

It is very evident to anyone seeing a group of animals on diets deficient in B complex, that they do not respond to the stimuli of different sounds, especially high shrill whistles. We have to determine the loss of hearing in animals on B deficiency diets, along modern lines. This hearing determination we hope to undertake very shortly.

The proof in humans of regeneration of the nerve in various parts of the speech area, and especially the nerve structure in the modiolus, as has been found in animals, may never be found. However, as time goes on, treated cases showing improvement in voice tests and audiograms may come to autopsy, with opportunity for histopathological studies that later on may be compared with case studies reported by Crowe[2] and his coworkers in their "Observations on the Pathology of High Tone Deafness." At the moment they have produced no evidence that the changes they so beauti-

fully describe may not have a definite nutritional deficiency background.

Perkins[19], discussing "Dietary Deficiencies and Certain Neurologic Syndomes," says, "since there are many diseases of the central nervous system in which the pathologic changes are degenerative instead of inflammatory, and the etiology is unknown, a more thorough investigation of the action of dietary deficiency on nerve tissue may bring to light at least one factor in the cause of the disease."

We submit, therefore, that the changes recorded on the audiograms of cases to be reported below, present sufficient evidence that the changes in the curves were obtained as the result of adding the missing food essentials, contained particularly in the vitamin B complex.

REPORT OF CASES

Case 1.—Paul L., age 22. Referred by Dr. L. Coblentz, December 10, 1935, with the following notes:

"Patient has complained of marked dizziness the past three years; attacks more frequent of late and the past week has had spells three times a day after meals. During the past year has lost fifteen pounds in weight. In some of the spells the vomiting has been projectile in character." Deafness in left ear noticed at age of nineteen years.

Blood pressure 120/72. Heart and lungs normal. Further laboratory studies showed a basal metabolic rate of minus 20. Blood examination: Hgb. 100%; R.B.C. 4,950,000; W. B. C. 5,550; Polys 63%; lymphs. 35%; trans. 3%; eos. 2%.

Patient is a linear type. Height, 5 ft. 10½ in. Weight, 154 pounds dressed. Has no allergic history and none exists in his family. Patient seldom uses alcohol and smokes about five cigarettes daily. Coffee, two to three cups daily. No quinine or aspirin.

Dietary History.—From infancy his dietary has been deficient in iron, and vitamins B, C and D. In his early life he had too much white bread, too little vegetables and fruits. No citrus fruits whatever. During the past year or so his diet has been better, owing to the fact that he thought he had a stomach ulcer. All his life he has had a great deal of milk, and in the past three or four years has been in the sun a great deal.

Auditory History.—In addition to comments by his physician as above, patient says when attacks of vertigo are present he has the sensation of the room revolving around him. Tinnitus is present; used to be high pitched in character, now more of a rumbling noise.

Examination of ear, nose and throat: Nothing abnormal. Eustachian tube, left side open. Lewis test for stapes fixation is minus. Rinne, plus. Hearing for whispered voice, 30 feet in both ears.

Treatment consisted of intramascular injections of vitamin B_1 solution, 150 Sherman units per cc. prepared by Lilly and Company, six days each week, thirty-

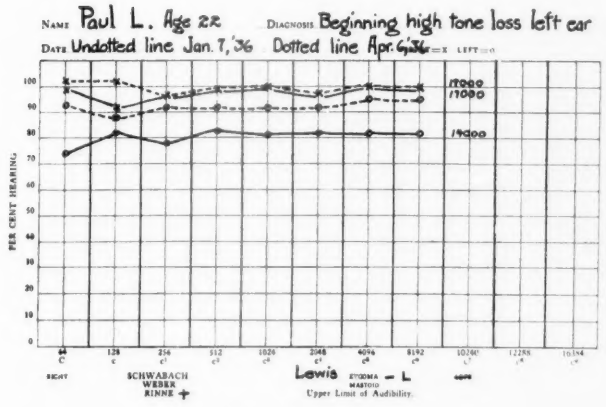

Fig. 1. Case 1.

eight injections in all, during which time he gained in weight. No vertigo, appetite improved. Associated and following each injection there was some flushing of the face, fullness and occasionally bad dreams. No other evidence of protein shock. On the termination of the injections another basal metabolic rate February 15th showed 9.8% minus. Pulse, 58-60. Weight, 150. February 20, 1936, patient given vitamin B1 complex made from rice polishings and furnished by the Vitab Company of Oakland, California; one dram three times a day, which is equivalent to 1,500 Sherman units daily. January 7, 1936, and April 4, 1936, his audiometric readings were as follows:

April 10th another basal metabolic rate showed minus 12.8%; pulse, 63-60. Weight, 156 pounds.

Comments: The interesting facts in this case are: First, the age of onset, with no evidence of other degenerative changes; second, the improvement in his basal rate and relief of vertigo and sensation of bodily good health following intramuscular injections of B1; third, the marked improvement in his hearing.

Further Comments: On April 20, 1936, the patient complained of an attack of vertigo and during the week three more. I saw him after one of these attacks and found him in a state of extreme anxiety and with a systolic pressure of 95. At this time I gave him Armour's concentrated cortical extract, thinking his attack might have been due to a low blood pressure. A day or two after, at the suggestion of a surgeon who had operated on him for appenditicis, he went under the care of another physician.

The question then ntaurally arose, was the patient a victim of an anxiety neurosis or the hyperventilation described by Dr. William Kerr of the University of California, in which type some of Doctor Furstenberg's cases may be classified, and that the early improvement in his vertigo might have been due to the acid character of the B1 solution (its pH being about 3). His audiometric curve had improved markedly and might have improved further on a longer continuance of the B complex or the filtrate portion, with or without producing further change in his basal rate on the addition of thyroid.

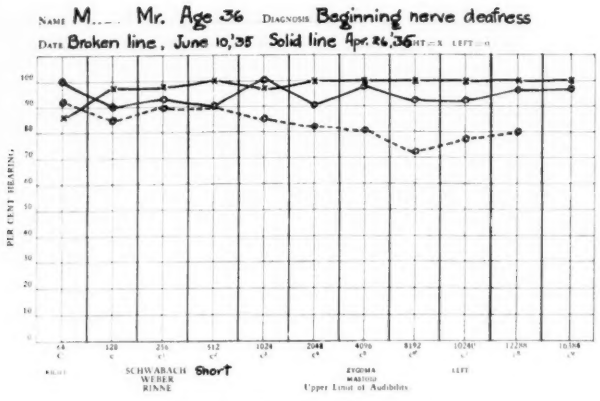

Fig. 2. Case 2. Graph A.

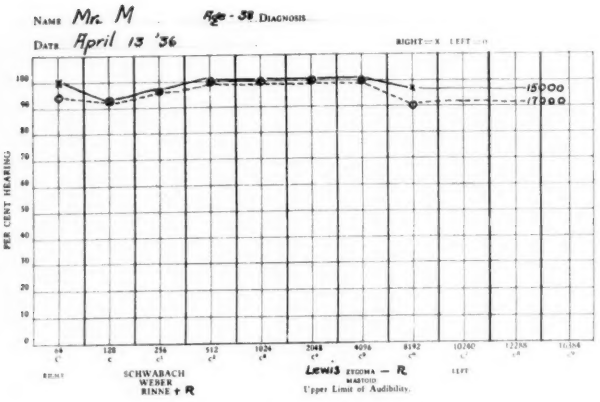

Fig. 3. Case 2. Graph B.

CASE 2.—L. E. M., age 38. Linear type; height, 5 ft. 9 in. Weight, 175 pounds. Consulted me April 26, 1935, complaining of high pitch tinnitus of one week's standing and some difficulty in hearing in the left ear.

Examination of the ears, nose, throat and eustachian tubes showed nothing abnormal, except that the posterior ends of both middle turbinates showed the white edema, described by Cody. His general health has always been good; has had the usual childhood diseases but no complications.

There is no hereditary deafness other than in the case of his father, who is seventy-eight years old and is getting deaf, probably of the high tone type.

His dietary history from William Watson's chart, plus a week's recording of food at each meal, suggests that he has been deficient in the quantity of B containing foods.

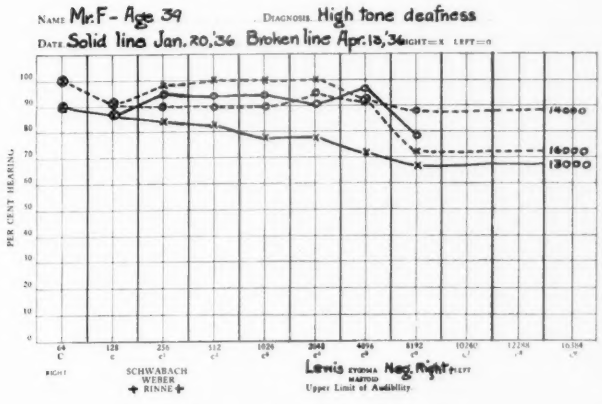

Fig. 4. Case 3. Graph A.

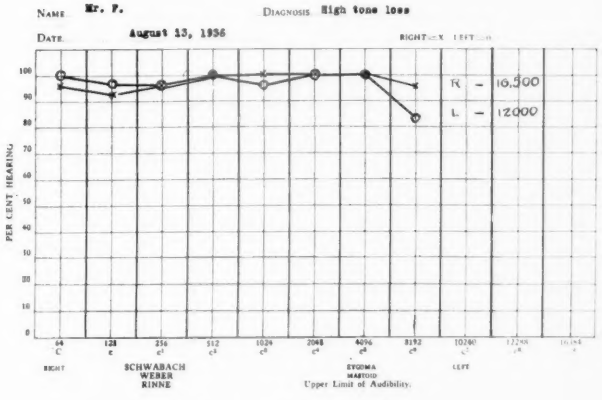

Fig. 5. Case 3. Graph B.

The improvement in his diet plus the taking of the B complex made from rice bran, one-half teaspoonful three times a day from April 29, 1935, to June 11, 1935, apparently brought about a marked change in the nerve as shown in audiogram No. 1. His B complex was continued from one month and then stopped.

Audiogram No. 2 taken April 13, 1936, shows a still further improvement. His high tone limit in the left ear April 29, 1935, was 12,200 cycles. April 13, 1936, it was 17,000.

Comments: This case shows that the additional use of the complex probably so affected the intestinal tract that absorption of B from the natural foods was made possible and is the explanation of his further improvement after stopping the B complex.

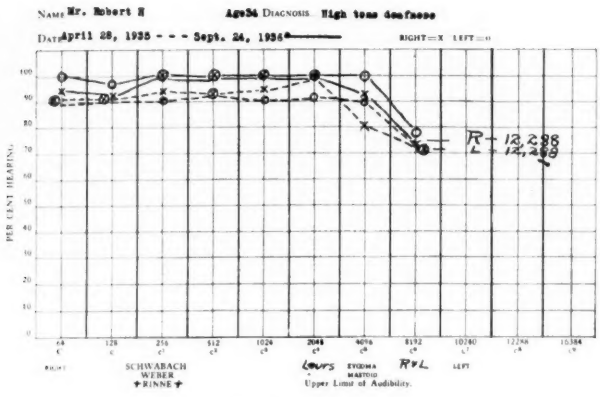

Fig. 6. Case 4.

CASE 3.—J. H. F., age 39. Linear type. Height 5 ft. 7½ in. Weight 160 pounds, consulted me January 20, 1936, complaining of stopped up ears and head noises. Nothing else wrong in his nose or throat. An audiogram was made January 21st and another April 13, 1936. His dietary study showed particularly that he consumed too much white flour products; did not eat enough eggs, milk, leafy vegetables and not enough citrus fruits.

As to treatment, he was given twenty-one intramuscular injections of B_1 solution, 180 Sherman units per cc furnished by Lilly and Company, which made him feel better and relieved him of most of his head noises. Unfortunately no audiogram was made when the B_1 was discontinued.

February 15, 1936, rice bran B complex was given, one dram three times a day (1200 Sherman units) and continued until April 13, 1936, when he was told to stop medication but to eat plenty of the essential foods, and to report in ninety days, for a hearing re-check.

The audiograms, Figs. 4 and 5, indicate the improvement of hearing.

CASE 4.—Robert H., age 35. Linear type. Height 5 ft. 10 in. Weight 155 pounds, consulted me March, 1935, complaining of frequent colds, several per year, always winding up with purulent discharge; never any bronchitis.

The report of his physical examination discloses nothing but a slight hypothyroidism for which he took thyroid for a period of time; also a mild type of constipation.

The x-ray pictures of his sinuses showed no evidence of pathology.

He said at times he thought there was a slight impairment of hearing. Examination of ear, nose, throat and eustachian tubes revealed no abnormality.

A dietary check with Watson's chart suggested a deficiency of vitamin A and B.

The photometer reading for A was normal.

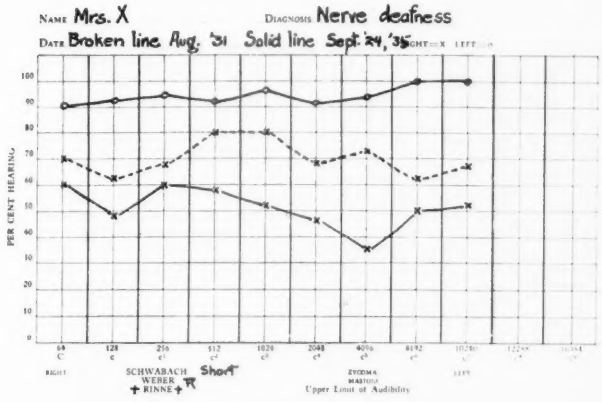

Fig. 7. Case 5.

Comments: With two months' treatment using first Burroughs and Wellcome Rhyzamin and after that California rice bran B complex, the audiometric curve between 64 and 4096 cycles became normal. Beyond that point there is no explanation for the failure for those tones to improve, unless one can attribute the loss above 12,228 cycles to a congenital defect or to some unknown infection.

CASE 5.—Mrs. X., age 36. Linear type. Height 5 ft., 6½ in. Weight, 140 pounds, consulted me first July 3, 1931. Her principal ear complaint was deafness in the right ear, attacks of vertigo, high pitched tinnitus, occurring very frequently during the past three years. Examination of ear and eustachian tube, normal. The outstanding thing in her general history is the large consumption of fluids daily, water, coffee, high-balls, amounting to 80-100 ounces daily; 20-40 cigarettes daily; not much extra salt. Added to this a feeling of general fatigue and apathy, associated with low blood pressure during attacks; and a lowered basal metabolic rate of minus 26%.

Her general medical check suggests that many of her complaints are built around a sex frigidity, though she has given birth to two children; but the fear of becoming pregnant along with this, and perhaps related to it, is a very perverse unwillingness to continue for any length of time methods of treatment advised by the several medical advisers she has consulted.

The restriction of fluids and salt, with ammonium chloride over a period of four weeks had a definite curative effect on her vertigo, though it did not improve her hearing above 5%.

From April 4, 1935, to Sept. 24, 1935, she took vitamin B complex and the difference in the two lines shows the improvement following its use. The taking of pituitary and thyroid at different times, in my opinion, had no influence on the hearing.

Comment: If she would have given up the use of tobacco and alcohol, and the excessive use of coffee, and taken the B complex

over a longer period of time, it is possible that her hearing might have improved still further. Unfortunately she belongs to the type so commonly seen among the deafened, who do not possess the ability to realize that the deafness has taken years to reach its height and that it cannot be improved in the course of only a few weeks, if at all!

CASE 6.—Mrs. W., age 35. Linear type. Height 5 ft., 10½ in. Weight 112 pounds, consulted me January 10, 1936.

Complaint: deafness; considerable difficulty in hearing when several people are present.

Further complaints: Marked night blindness. Good general health except frequent colds from childhood until favorably influenced in recent years by Haliver Oil each fall. Had measles, mumps, chicken pox; occasionally uses aspirin; no quinine; coffee moderately; no tea, tobacco, alcohol. Tooth history good.

Laboratory studies: Basal metabolic rate was minus 22 percent. Blood count normal. Hgb. 80 percent. Fasting Blood Sugar: normal.

Vitamins: *A*: In spite of the fact she apparently had an adequate amount of fats, she undoubtedly has been rather markedly low in A all her life. Her history of many colds in the early years of her life and particularly the night blindness indicate it. In fact a recent test with the visual photometer gives a minus 3 reading.

B: low all her life. *C*: deficient all her life until the last few years. D: probably low most of her life. *G*: adequate.

Her prenatal diet was low in B, C and D and iron.

RESUMÉ OF DIETARY HISTORY

	Carbohydrate	Protein	Fat	Iron	Calc.	Phos.
Infancy				Def.	A	A
Childhood	High	A	A	D	A	A
Adolescent	A	A	A	D	A	A
Adult	A	A	A	D	A	A

Key: A is adequate; L is low; D is deficient; ? is questionable.

Ear History: First observed hearing difficulty about age of 27 years. There was no accompanying tinnitus or vertigo. Some difficulty in hearing over phone in one ear. The principal trouble was in hearing conversation, especially when several people were talking.

Examination of ears, nose and throat, eustachian tube normal. The posterior ends of the middle turbinates were white: edema of Cody (with pharyngoscope).

Hearing test: R. ear—Whisper, 9 feet. Conversation, 24 feet.
L. ear—Whisper, 18 feet. Conversation, 15 feet.

Rinné: plus. Schwabach: short. Lewis fixation: negative.

Treatment: Patient was given B_1B_2 (Lilly and Company) 1200 units daily, beginning January 10th and continued same until July, 1935, at which time the B complex was added. April 9, 1936, Squibb's "Navitol" one capsule t. i. d. added on account of low photometer reading.

The accompanying audiograms show the original hearing, and the hearing on June 22, 1936.

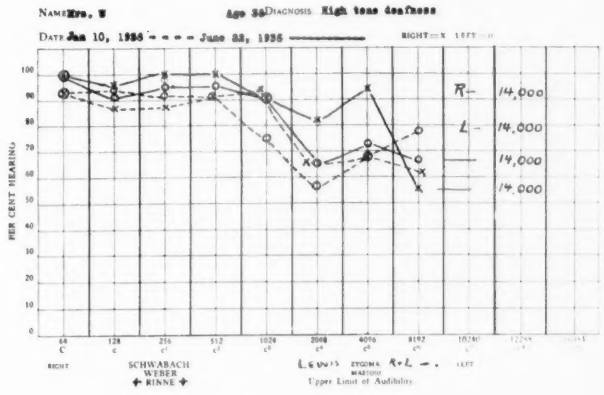

Fig. 8. Case 6.

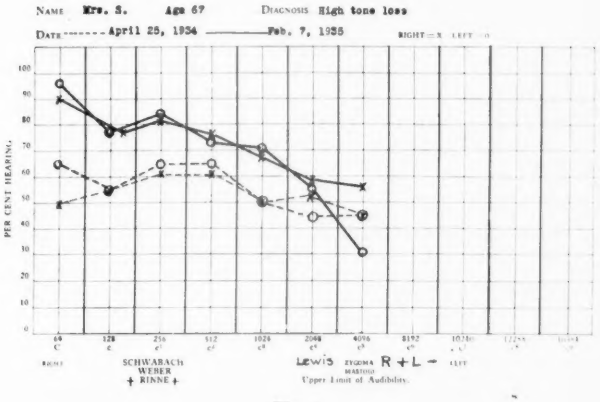
Name Mrs. S. Age 67 Diagnosis High tone loss
Date - - - - - April 25, 1934 ——— Feb. 7, 1935
Right = X Left = O
PER CENT HEARING
SCHWABACH
WEBER
+ RINNE +
Lewis Zygoma Mastoid R+L −
Upper Limit of Audibility

Fig. 9.

Comments: To me this case is particularly interesting because of the possibility of a hereditary factor. Her mother, now 68 years of age, gave a history of deafness first noticed at the age of 31, following the birth of her first child. She evidently had some ductless gland disturbance at puberty. At the age of 60 years she had a thyroidectomy. She also had a bad nutritional history most of her life. In the summary of her dietary history, iron was apparently low from infancy to later adult life. Vitamin B low all her life; C low until later adult life; D deficiency in infancy and low since then. Her history of chalky teeth in childhood, all filled early, supports the idea of nutritional deficiencies.

The curve on April, 1934 (Fig. 9), with the history of familial deafness and of her own deafness beginning after the birth of her first child might be interpreted as otosclerosis, and yet the rise of the curve, especially as to the low tones following treatment, is sufficient evidence of a nerve involvement from the beginning. This idea is borne out by the fact that in no case of otosclerosis have I seen such a marked rise in the low tones.

Case 7.—Mrs. C., age 51. Linear type. Height 5 feet 9 inches. Weight 175 pounds, consulted me January 17, 1936, complaining of difficulty in hearing when several people were talking. Onset was gradual, over a period of several years. Not subject to colds. No hay fever. No migraine. Has had colitis over a long period; low pulse and low blood pressure (105-78) and fatigue. Has been taking thyroid off and on for three to four years.

Nutritional disturbances play a prominent part in her family. An older sister suffers from extreme overweight, thyroid disturbance, gall bladder trouble, nervousness. Her father was heavy and had arthritis. A brother, sister, and the patient all had constipation. The resumé of patient's dietary history is as follows:

	COH	Protein	Fat	Fe	Calc.	Phos.	Vit. A	B	C	D	G
Infancy	It is safe to assume that Fe and vitamin C were deficient										
Childhood	A	L	A	L	D	L	A	D	L?	A	L?
Adolescent	A	L	A	L	D	L	A	D	L?	?	L?
Adult	A	A	A	A	D?	L	L?	D	L?	L	A
Past 3 years	A	A	A	A	L?	L	A	D	A	L	A

Key: A is adequate; L is low; D is deficient; ? is questionable.

Examination of nose shows a large perforation following typhoid. No evidence of sinus disease, though she had a slight whitish postnasal discharge. Left antrum opened intranasally for pain and headaches. Right ear normal, though the anterior wall of the external canal bulges and prevents seeing the lower half of the light spot. Left mastoidectomy in 1924.

This patient was given thirty injections of Lilly's B_1 solution parenterally. Not much change, if any, followed the injection. After the injections she was given absorbate capsules, made from California rice bran—B complex, containing 300 I. U. of B_1 and 75 I. U. of B_2. These capsules were taken until June, 1936, when she was given the entire complex.

Treatment was discontinued August 18, 1936, for a three months' period.

Comment: The interesting part of this case is the fact that while she had taken thyroid, calcium, etc., for quite a period before consulting me, and continued the same from January to August, 1936, at no time did it appear to influence the nerve, and the improvement in the audiogram curve did not follow until the B factor was added.

Case 8.—Dan F., age 54. Linear type. Height 6 feet. Weight 185 pounds. Consulted me January 15, 1936. Said he first noticed his deafness in 1915. Thought that perhaps it might have been due to working in a stamp mill during his youth. There were no associated colds or tinnitus; nothing was found in his ears, nose or throat. He is confused when several people are talking.

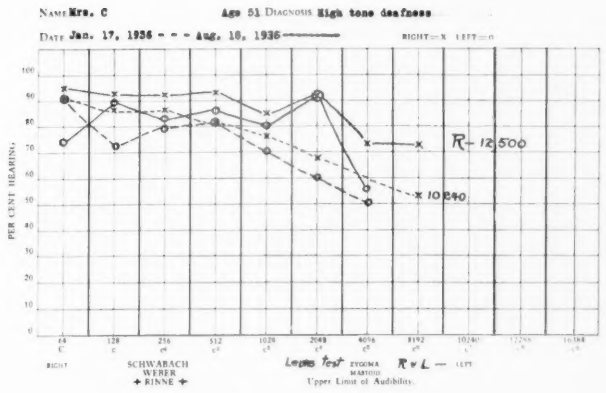

Fig. 10. Case 7.

Does not use tobacco, alcohol sparingly, coffee two cups daily, uses no drugs. Had scarlet fever at four years of age; mumps later; no sequellae; no deafness in the family. Blood pressure is low. Basal rate is minus 15.2 percent. Except for fatigue, general health is good.

A rather complete dietary history does not give much information about his feeding in infancy. He weighed twelve pounds at birth and was breast fed. His childhood dietary history shows he was deficient in vitamin B. He did not drink much milk, because he did not care for it. He was born on a ranch in Southern California, where there were cows, citrus fruits, etc. He ate plenty of eggs and cream. Had plenty of sunshine. His only source of vitamin B was oatmeal porridge daily, and beans once a week, with some fruit between meals. His source of vitamins A, C and D was apparently sufficient.

In his adult period the vitamin B inadequacy carried over from childhood; also his vitamin C intake is low during some seasons of the year. The same applies to vitamin D, as he lives in the Bay region. He has had a high carbohydrate intake all his life and this, coupled with his low vitamins may explain to some extent the fatigue he has complained of during the past ten years. While the lowered basal rate may be related to his thyroid, Bogert seems to think it may be fostered by the low vitamin B intake.

The patient was, therefore, given a series of intramuscular injections of vitamin B_1 (Lilly and Company) from January 23, 1936, to March 4, 1936. From March 4th to June 15, 1936, he was given capsules of B_1B_2 (Vitab Company) 900 International Units B_1 and 75 of the B_2 units. This was changed to the entire complex and is being taken at the present time, October, 1936.

Comments: On the audiogram the broken lines are of the October 8th, 1936, hearing check. I have no explanation at the moment why the tone 4096 shows a drop of over 20 percent on that date, whereas on previous tests the reading was higher.

What influence noise has on a nerve with a resistance lowered possibly by a B deficiency is not at the moment clear. Some of his

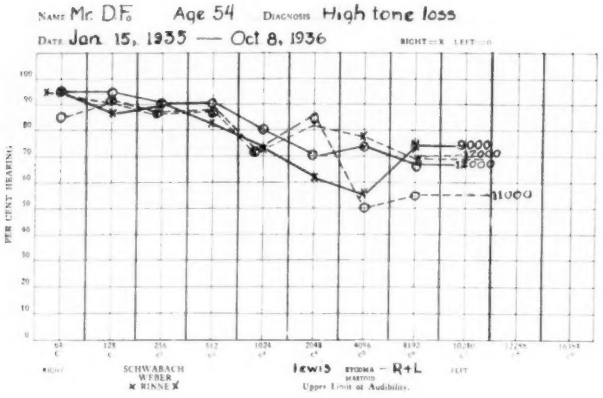

Fig. 11. Case 8.

notes in the hearing area have improved. If, as time goes on, there is still further improvement, it will then be rather definite that the loss in the high tones is related to his deficiency and not to the noise of the stamp mill in his earlier life.

Case 9.—Mrs. S. E. W., age 54. Height 5 feet. Weight 105 pounds. Consulted me first in 1919 for hay fever of the spring type. April 5, 1929, she again consulted me for a slight difficulty in hearing, noticed when several people were talking together. An audiogram and other tuning fork tests were made which showed that the hearing difficulty was of the perceptive type.

General History: The patient gave a long history of gastrointestinal difficulties; a suspicion of duodenal stricture; spastic colon; constipation, etc. She had a low blood pressure and her basal rate was minus 22 percent.

A childhood dietary history showed a high intake of cereals, potatoes and sweets. Her first teeth decayed early but her permanent teeth appear good.

Much of her adult life has been spent traveling, big game hunting, camping and cruising with her husband in Alaskan waters, and during these periods the foods were largely canned and necessarily the protein intake and the carbohydrate intake in relation to fats were low.

Her vitamin A, from the photometer test, is slightly deficient. B and G, due to the intestinal condition, apparently have not been properly absorbed, hence must be considered as low. C is also apparently low, especially during the summer months.

Examination of ears, nose and throat showed nothing abnormal. Tonsils and adenoids removed at the age of sixteen years.

Recheck of the hearing in December, 1935, showed some improvement over that of April 29th; the cause was unexplained.

Treatment: On January 6, 1936, a series of injections of Lilly's B_1 solution (150 Sherman units per cc.) was started. Thirty-five injections in all were given

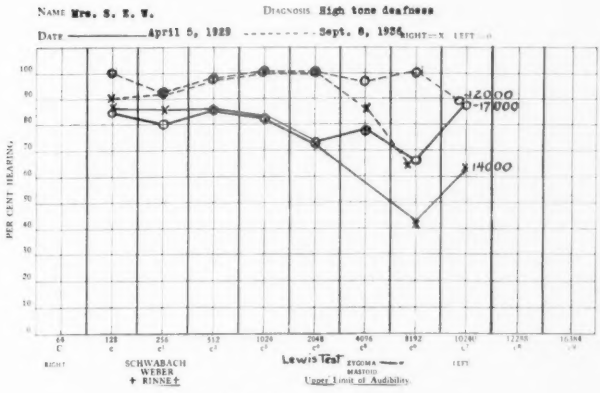

Fig. 12. Case 9.

daily. Following the first six or eight injections there was a feeling of congestion in the head with headache, lasting an hour or two. There was no other evidence of protein shock. Within two weeks the effects of the injections showed a more natural bowel action, increase of appetite and a feeling of general well being.

As to the ears, there was possibly a slight improvement in tones of 128 cycles and 1024 cycles in the left ear. No change was demonstrable in the right ear.

At this time she was started on B complex made from California rice polishings and has been taking the complex to date (November, 1936).

In May, 1936, she was put on two grains of Burrough and Wellcome thyroid extract and is still on this dose, although the basal metabolic rate is now within normal range.

Comment: This case is reported because she shows several conditions aside from the high tone loss, i. e., allergy, marked gastrointestinal conditions, low metabolic rate and vitamin deficiencies.

The especially striking fact in this case is the complete relief from hay fever, the first since 1919, even though she spent a part of the hay fever season at her residence near San Francisco, and the rest of the season traveling through Glacier National Park, Yellowstone National Park and back to her home, all during the grass pollenizing season. During all this period she continued taking her B complex.

In view of McCarrison's remarks, at the recent meeting of the British Medical Association's Section of Nutrition, that vitamin B has an influence on the autonomic nervous system and also from the fact that several of my own cases of perennial hay fever have improved in rather an astonishing way, one can almost make the

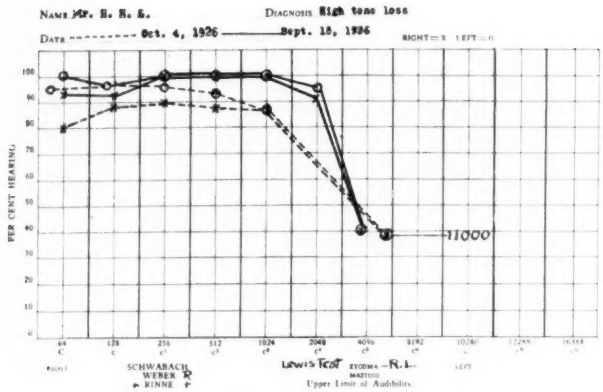

Fig. 13. Case 10.

claim that some fraction of the B complex has an influence on these conditions:

Case 10.—Mr. H. H. S., present age, 54 years. First consulted me October 4, 1926 (age 44), stating that he had been hard of hearing for several years. At that time he had an exostosis in the right external canal, otherwise nothing abnormal. He had infected tonsils. He had had several teeth removed six months before I saw him.

An audiogram made at that time showed a high tone limit in the left ear of 11,000 d.v. The right ear high limit was 4096 d.v., but with a loss of 60 percent.

No further attention was paid to his hearing until November 6, 1935, when another audiogram was made and in January, 1936, a careful physical study was made by Doctor Friedlander and also a nutritional study was made.

The report of the former says under "impression": "no organic disease, but a slight suspicion of cholecystitis. To have x-ray of left lower crowned tooth for abscess. Should have a basal."

NUTRITIONAL HISTORY

	COH	Protein	Fat	Fe	Calc.	Phos.	Vit. A	B	C	D	G
Infancy	A	A	A	D	A	A	A	D	D	D	A
Childhood	H	A	A	D	A	A	A	D	D	D	A
Adolescence	H	A	A	D	G?	A?	A	D	D	D	A
Adult	A	A	A	G	L	G	G	D	D	F	A

Key: A is adequate; G is good; L is low; H is high; D is deficient; F is fair.

Dietary Suggestions: Milk or cheese is to be taken to improve his calcium phosphate intake; Sperry's wheat hearts; Ralston's cereal or shredded wheat; whole wheat bread in place of white bread; B_1 concentrate to give more vitamin B. Orange juice, fresh fruits; cole slaw or lettuce to be eaten to increase the vitamin

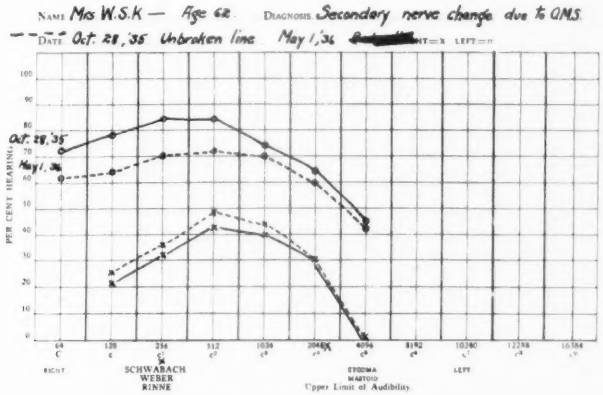

Fig. 14. Case 11.

C. This diet, together with liberal doses of the B complex, half an ounce three times a day, has improved his sense of well being and has had a definite influence on his gain in hearing.

Comment: This patient shows from his audiograms a slowly progressive high tone loss over a period of twenty years, with his greatest hearing difficulty in a room where several people are talking. The finding of the loss of all high tones above 4096 cycles and only 60% of that tone lost, at the age of forty-four years, suggests a possibility that it is perhaps associated with some unknown infective process on top of a nutritional deficiency.

CASE 11.—Mrs. W. S. K., age 62. Height 5 feet 6 inches. Weight 180 pounds, a pituitary type. Has been treated at intervals for a recurrent mucopurulent discharge from both ears, having its beginning in an acute infection many years ago. Perforations in the drums had never healed; each attack was accompanied by a dermatitis of both concha, red and scaly at the beginning of each attack and the discharge was watery in character.

In October, 1935, her physician noticed some difficulty in her ability to hear ordinary conversation and referred her to me.

No general or nutritional studies were made.

She was given the B complex partly to see what effect it would have on the eczema, without much hope that the audiogram curve would be influenced.

In spite of the fact that the complex was taken indifferently, the curve shows improvement on both ends of the scale, especially the low side. Her hearing for speech improved to such an extent that she could hear moderate conversation at six feet. The patient uses forty cigarettes daily. If smoking were stopped and she would take the complex regularly, there might be hope for further improvement on the high side of the tone scale.

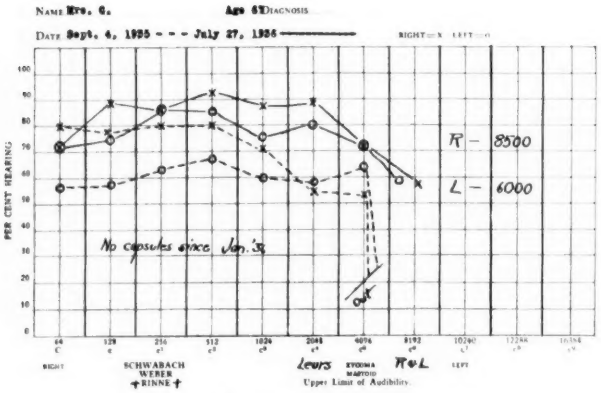
Name Mrs. G. Age 67 Diagnosis
Date Sept. 4, 1935 - - - July 27, 1936 ——— RIGHT = X LEFT = O
PER CENT HEARING
R - 8500
L - 6000
No capsules since Jan. '36
SCHWABACH
WEBER
RINNE
Upper Limit of Audibility

Fig. 15. Case 12.

Case 12.—Mrs. J. J. G., age 67, consulted me for defective hearing in 1930, more noticeable when a number of people were talking. Each year since there has been a slow but gradual loss in the entire tone scale.

She has been under the observation of a general physician for years and her diet has been well chosen.

She was given Furstenberg's low salt diet plus large doses of ammonia for some time with some apparent gain in her hearing, but it had to be discontinued owing to stomach upset. She was given Extract Vitamin B (Parke, Davis and Company) over a period of time with definite improvement.

When the audiogram was made, July 27, 1936, she had taken the remedy for at least three months, suggesting the idea that the intestine was absorbing a normal amount of B_1 from her diet.

Case 13.—Mr. Thos. B., age 72. Linear type. Height 5 feet 10 inches. Weight 170 pounds. Consulted me first about his hearing April 23, 1929, stating he had difficulty in hearing, especially when several people were talking. There was nothing abnormal in his ears, nose or eustachian tubes. He had had fairly large infected tonsils that were removed fourteen years ago. His Rinne test was positive. Schwabach short. There was no history of deafness in the family.

In his general history during the past twenty years there has been nothing unusual beyond arthritic complaints; some gastrointestinal upsets; constipation and colitis especially in the past few years. His dietary history most of his life has been one of good living except for the use of considerable white flour products; not much milk or whole wheat products. He has been deficient in vitamin B for a long time.

He was given, at that time, a diet low in salt and an attempt was made to give him ammonium chloride in large doses, but as it upset his stomach and frequently made him lose his meals, it was abandoned. Vitamin B complex was then given with the result as shown in the following audiogram.

Case 14.—F. E. B., present age (1936), 75 years. Seen first September 13, 1926, at which time he complained of some difficulty in hearing. At the age of

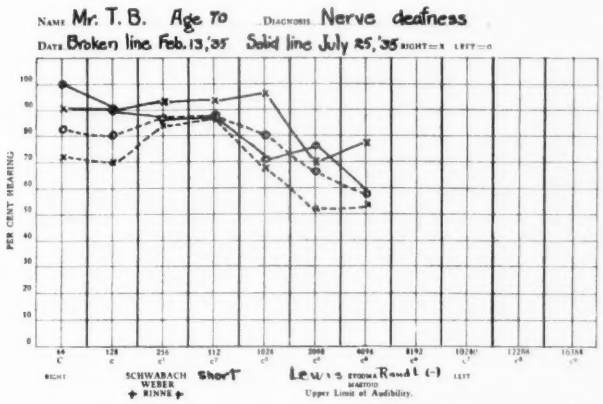
NAME Mr. T. B. Age 70 DIAGNOSIS Nerve deafness
DATE Broken line Feb. 13, '35 Solid line July 25, '35 RIGHT = X LEFT = O
PER CENT HEARING
SCHWABACH Short
WEBER
+ RINNE +
Lewis stroma Rand (-)
MASTOID
Upper Limit of Audibility.

Fig. 16. Case 13.

33 years he had an abscess in the left ear that discharged for several years, for which his ossicles were removed. The ear continued to discharge for several years and finally stopped.

During 1930 he developed an infection in the left ear that discharged very freely and finally he developed signs of perilabyrinthitis with nystagmus, dizziness and disturbances of equilibrium. On July 30, 1930, a radical mastoid operation was performed; a large mass of granulations and cholesteatoma was found in the antrum and middle ear.

May 1st and August 2, 1931, audiograms were made showing that the hearing in the left ear was practically gone. Following this period his hearing for voice became very bad and frequently one had to speak very loud. He heard with the greatest difficulty. He tried various hearing aids without any help.

In February, 1935, I put him on Furstenberg's low salt diet, restricted his fluid intake and gave him large doses of ammonia chloride for several weeks with some improvement of an instability he had noticed when walking. There was, too, a slight improvement in his hearing curve.

March 12, 1935, I began the use of the B complex, commencing with half a drachm after meals and increased the dose to one drachm three times a day. Along in September, 1935, I added Carotene, 30,000 units daily, which was kept up for several months.

Also, he had been taking thyroid off and on since 1932 when his physician found his basal metabolic rate low. In April, 1936, his visual photometer was found low in spite of the Carotene given.

Comments: This case is very illuminating, first, because the patient shows a complete loss of function of the left cochlear apparatus, depending probably on secondary infection of the nerve, following a radical mastoidectomy, and second, the development of

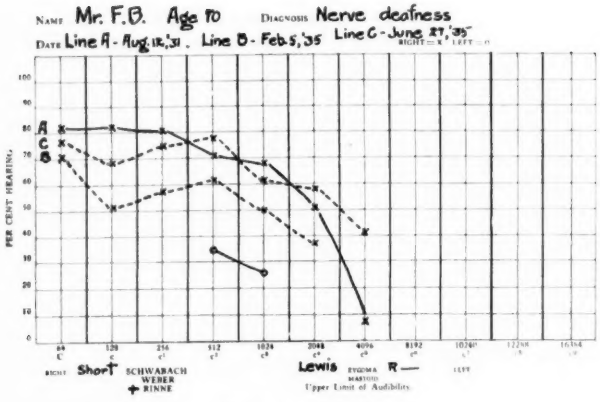

Fig. 17. Case 14.

useful hearing following the use of the B complex; all occurring in a man aged seventy-two years. This is evidence enough that help should be offered at any age.

CONCLUSIONS

1. That the work of Professor Peters of Oxford proves conclusively that some factor in the vitamin B complex is necessary for perfect biologic equilibrium, i. e., the oxidative reduction system, in order to prevent the accumulation of lactic acid in certain parts of the brain.

2. That the finding of a definite demyelinization in both branches of the eighth nerve in a series of rats and chicks on a diet deficient in the different factors of the vitamin B complex, B_1B_2, and especially the filtrate factors B_6B_7 or K_1K_2 as shown in Dr. W. P. Covell's histopathologic studies, warrants the claim that the lack of sufficient B in the human diet is probably the principal cause of the degeneration found in the nerve.

3. That the clinical use of B_1 parentally, B_1B_2 absorbate, and particularly the rice bran B complex, appears to be the cause of the improved hearing noted in the audiograms as shown in the reports of clinical cases.

4. That large doses of carotene (30,000 units daily), over a period of months, has caused no further change in hearing as demonstrated by the audiograms. Perhaps if the dosage of carotene or vitamin A was carried on over a longer period under the control of the photometer, further changes might be noted.

5. That the use of thyroid, except possibly in rare cases, does not influence the nerve changes. Its effect probably is to help in the general metabolic processes.

6. That the lowered basal rates in many instances are related to nutritional deficiencies.

7. That Cody's finding of a whitish edema in his animals on a B deficiency diet, and as seen in humans with the pharyngoscope, appears to be somewhat conclusive as to vitamin B deficiency.

8. That no interpretation can be made regarding the finding of high tone deafness in the linear type of body build.

9. That a report on the use of varying strengths of the filtrate factors, with and without the B complex, in high tone loss, as well as in otosclerosis, will be presented at a later date.

FITZHUGH BLDG.

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IX

DIPLACUSIS BINAURALIS DYSHARMONICA

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Of the resonator theory of hearing much has been written pro and con. Transient alterations in pitch resulting from inflammations of the middle ear can best be explained by this theory. Many references to this condition are to be found in the literature but having, myself, been the victim of an attack, I was able to study it and make records of sufficient accuracy to warrant a description, together with what seems to me to be a logical, if not a demonstrable, theory.

The phenomenon under discussion occurred as the result of an extremely acute middle ear infection. In the midst of a severe purulent rhinitis, pain was suddenly experienced in one ear, the right. Within three hours the drum ruptured spontaneously, liberating a large amount of serosanguineous fluid which contained pneumococci (type III). A copious discharge of this fluid continued for three days when it ceased rather abruptly without any gross alterations in its character having occurred. The hearing on the affected side was reduced about fifteen units throughout the tone range. The pitch, as perceived by the diseased ear, was raised from one-quarter tone to four and a half tones at various frequencies.

Tests made with tuning forks and with two audiometers (one at each ear) showed the discrepancy in pitch perception to begin at 1,024 vibrations and to increase with the elevation of pitch, reaching a maximum difference of four and a half tones at the limit of hearing in the C^7 octave. The two audiometers (one a 1A Western Electric, the other a laboratory instrument with a continuous pitch range) were made to oscillate in exact unison, as nearly as could be determined by listening to the two receivers with the normal ear and eliminating beats. At the various pitch levels, sounds interpreted by the normal ear as C-natural, were heard by the affected ear as C-sharp, D, D-sharp, E, F, and F-sharp respectively. (Fig. 1.)

The condition persisted unchanged for twelve days, after which it returned to normal within seventy-two hours. The improvement progressed uniformly along the tone range; that is to say, the higher

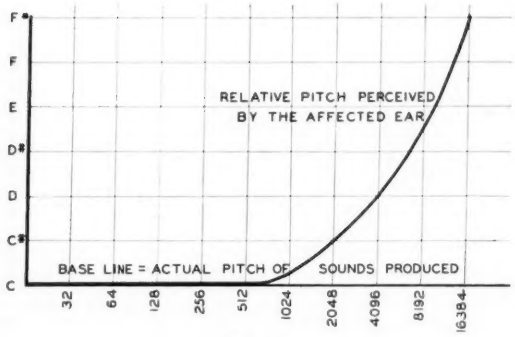

Fig. 1

ranges having deviated farthest from the normal, were the last to return to it.

Macfarlan[1] described a case of diplacusis which he himself experienced during an acute attack of purulent rhinitis. Shambaugh and Knudsen[2] have reported others. They attribute the condition to "some pathological deposit adhering to the tectorial membrane: a blood clot or a fibrinous deposit" which causes a given segment of the tectorial membrane to resonate to a tone above or below the usual one. Macfarlan concludes that the exact pathological condition is still conjectural.

Politzer[1] (1894) reviewed the symptom briefly and the following are excerpts from his text:

"The qualitative change in the perception of hearing which is most frequently manifested in a false perception of pitch should not always be considered a symptom of a disease of the auditory nerve, for, as we have seen, there may be variations in pitch of one-fourth to one-half tone due to the changes in tension in the sound-conducting apparatus during middle ear affections. Greater differences in perception to the next higher third (Moos) or 0 to 2 tones higher or lower (Knapp, Swan, Burnett, Pomeroy, Gruber) are to be considered due to the auditory nerve apparatus being out of tune, and they are specially painful and disturbing in musicians on account of the subjective dissonance in unilateral affections. The difference in pitch of sounds heard by the two ears through air conduction only rarely signifies a similar condition for bone conduction (O. Wolf).

"The want of tune of one ear is to be classified with the rarely occurring diplacusis (Paracusis dipl.), an anomaly of perception in which either each sound or only certain tones are heard twice. I have observed this disturbance of hearing several times with otitis media acuta, with serous middle ear catarrh, and with chronic middle ear suppuration. . . .

"Barth considers the diplacusis binauralis to be principally due to the middle ear, and that the diseased ear hears no other tone than the normal one, but with a

different pitch. Bressler reported an interesting case of double hearing, which Gumpert observed on himself. After a protracted inflammatory ear affection double hearing occurred. He heard spoken words as though they came from two different directions. Both words were perceived without an interval between, the second not being an echo of the first. He was only able to distinguish between them by the difference of their tone. That he did not hear double with the diseased ear was distinctly proved by hermetically stopping the meatus of the sound ear.

"Kayser (Berl. Congr., 1890) differentiates a diplacusis dysharmonica and echoica. By the first, two tones are perceived, the diseased ear hearing a higher or lower tone than the normal one. By the second form, as described by me above, the sound is heard later and weaker, the same as an echo.

"Jacobson disputes Barth's explanation of double hearing, and considers with Wittich that the elastic end apparatus of the auditory nerve is totally or partially out of tune, therefore there is a participation of the labyrinth with the middle ear affection. . . ."

I agree with Jacobson and Wittich that it is the elastic end apparatus of the auditory nerve which is affected and not the middle ear, as Barth contends.

That there should be some impediment to the amplitude of sound transmisison by the conduction apparatus is obvious. The change in pitch, however, must be the result of a disarrangement of the perception apparatus since the actual frequency of the incoming sound wave cannot be altered in the middle ear. The change in pitch perception is confined also to the upper ranges—those associated with the fibers of the lower cochlear whorl involved in the tympanic inflammation.

Accepting, for the moment, the resonator theory of sound perception and resorting to an analogous mechanism, the pitch deviation may possibly be explained as follows:

If a C sound be produced near an open-strung piano, the C string will vibrate. An observer watching this string, and following its connection with a certain white piano key, may thus identify the original sound as C. Similarly C-sharp, D and the rest of the scale. Here the strings are the resonator or cochlea, the piano action represents the communicating line, or eighth nerve; the piano key, together with the observer, constitutes the brain. (Fig. 2.)

If for some reason the end of the piano were to swell or warp in such a way that the tension of the strings were slightly relaxed (dotted line, Fig. 2) the C string would now sound B; C-sharp would slip to C; D to C-sharp and so on.

The experiment of resonation is now repeated; the C sound is produced. The string which now responds is the erstwhile C-sharp

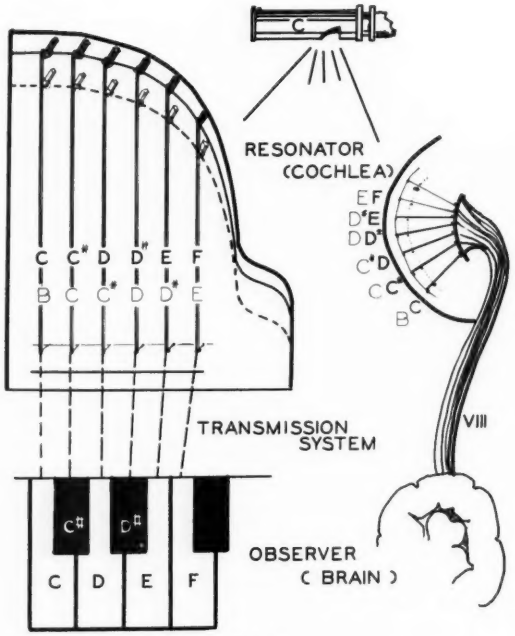

Fig. 2

string which is now tuned C. The observer again traces the mechanism related to the vibrating string. This time it leads him, not to the white C key but to another slightly higher in the range: a black C-sharp. Unless he has "absolute pitch" he is now under the impression that the exciting sound must be C-sharp, whereas it is the same C which caused the C string, associated with the white key, to respond when the piano was in tune.

By converting this mechanism into terms of cochlea, auditory nerve and brain center, the augmented pitch perception may possibly be explained. Confirmatory evidence may lie in the fact that as the pitch rises, the discrepancy increases. According to the laws relating to vibrating strings, the frequency of vibration is logarythmically proportional to the tension. In terms of the piano again, if all the strings were relaxed by moving their pegs an equal distance, the pitches of the high, short strings would be reduced to a proportionally greater extent than the longer strings of the middle and lower ranges. If, therefore, an even swelling occurred throughout the affected portion of the cochlea, and the cochlea were indeed a resonator, it could

be predicted that the higher pitches should deviate farther than the lower, which was actually the case.

It is not here intended by the employment of the piano string analogy to imply that that change in the cochlea was necessarily one of length. Frequency of vibration is equally a function of mass and tension according to the formula for vibrating strings:

$$n = \frac{1}{l}\sqrt{\frac{t}{m}} \quad ;$$

n being the number of vibrations per second; l the length in centimeters; t tension in dynes; and m the weight of 1 cm. of the string in grams. Any of these factors may be altered by an inflammatory process.

It has been suggested that the elevation in pitch was the result of the increased amplitude required to reach the threshold of hearing in the affected ear. I cannot accept this explanation, in as much as the same sound could be heard simultaneously in both ears at two pitches. At a symphony concert, for instance, the violins were badly out of tune with the basses, for the affected ear, although the sound obviously reached both ears with the same intensity. Furthermore, if the diplacusis were a function of intensity it would be a common phenomenon in acute unilateral middle ear infections.

Unfortunately, bone conduction via the vertex with the audiometer was not recorded.

Within the last week I have seen another case of diplacusis in which the pitch of the affected ear was lowered instead of raised and in which the disparity increased as it approached the lower end of the scale. This symptom lasted only a few hours and disappeared before the two audiometers could be arranged.

Since this article went to press, I have come across an account of a similar explanation offered by G. E. Shambaugh, Jr., in a case of low tone diplacusis published two years ago.[4]

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X

A METHOD OF STUDY OF THE NASAL MUCOSA IN RELATION TO THE TUBERCLE BACILLUS*

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EL PASO, TEXAS

Certain methods of demonstrating organisms in the nasal mucous membranes, such as microscopic examination of smears from swabbings, tissue sections or cultures and animal inoculations, have been used by the majority of investigators in the field of nasal bacteriology; however, as far as the literature suggests, the use of epithelial scrapings in demonstrating organisms in the nasal mucosa has been employed infrequently, if at all. In order to bring this method to the forefront it is necessary to focus attention on some one organism. I have chosen the bacillus tuberculosis for this study because of the characteristic staining method of the bacillus (acid-fast), and subsequent ease of classification. The purpose of this paper, therefore, is to show the existence and incidence of tubercle bacilli in the subepithelial structures of the nasal mucous membranes obtained by epithelial scrapings.

In finding tubercle bacilli, the removal of the ciliated surface epithelium offers certain advantages over other methods of searching the nose for organisms. This method of superficial scraping is feasible by reason of the fact that the surface epithelium is very loosely attached to the basement membrane. In this statement I am corroborated by such an investigator as Ross.[26] The procedure obviates the necessity of removing a section or gross amount of tissue, and is quickly and easily accomplished. The organisms are found with greater facility after they become enmeshed in the cilia and deeper structures of the epithelium than in the nasal secretions, because these, together with the other débris, are ever changing; they may contain organisms at one time and be free of them at others. Again, the removal of a flat surface of the epithelium offers a greater possibility for the gathering of bacilli than in a small, punched out biopsy with cross sections made showing little of the surface structure. In the former method the opportunity of studying a greater

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area of the nasal mucosa is similarly allowed. In a case of frank nasal tuberculosis this fact may explain the reason why the organisms are so rarely discovered in the tissue obtained by the usual biopsy and sectioned by the usual methods.

Therefore, if it can be demonstrated that tubercle bacilli are found in the nose, and especially sub-epithelially in the nasal mucosa, these cases may be considered as potential cases of nasal tuberculosis. Twenty years ago considerable rhinologic interest was manifested in phthisis, but since that time comparatively little attention to the subject is to be noted. It is hoped that the following experimental findings may afford added insight into the knowledge of nasal bacteriology and may further stimulate the interest in nasal tuberculosis.

When one reviews the literature of nasal tuberculosis, the pathology, the histopathology and the laboratory findings, he is at once impressed not only by the dearth of recent articles pertaining to the subject, but by the rarity of tuberculous lesions wherein tubercle bacilli can be found culturally, microscopically in smears, through biopsy, or by animal inoculation with any degree of constancy. This is verified in case reports by such investigators as Russo-Fratassi,[25] Zimmer-Leibovici,[37] Kistner[12] and Freer.[6]

Such writers as Renshaw,[24] Cornet,[5] Spencer[28] and Wright[33] have demonstrated that tubercle bacilli can penetrate the unbroken mucous membrane, and having lodged in the subepithelial structures may there produce various types of tuberculous lesions, or, as is usually the case, they may be destroyed in the tissue without producing local or general symptoms. However, the organisms may pass through this portal of entry, lodge in the regional lymph nodes and, in turn, this point may become the focus of infection, giving rise to tuberculous activity elsewhere in the body.

It is conceded that tuberculosis of the nasal mucosa occurs primarily as a result of infection introduced from without and secondarily from within. The method of infection of the nasal mucous membranes may be divided, therefore, into ectogenous and endogenous types of invasion. In the ectogenous form the inoculation with the bacilli is produced by trauma where it may follow operations on the nose. Another portal of entry may be through abrasions made by the finger or handkerchief. Though the entrance of the bacilli into the mucous membranes may not result in a tuberculous lesion in the nasal mucous membrane proper, entrance may thus be gained into the regional lymph nodes and the nose be the seat of but a transient infection.

Considerable numbers of tubercle bacilli must occasionally be drawn into the nasal cavities in healthy individuals. That the nasal secretions of a considerable percentage of healthy individuals contain tubercle bacilli has been shown by numerous investigators such as Cobb and Nagle,[4] Massione[16] and Zancla.[36] In still greater percentage must these organisms be present in the nasal cavities of the tuberculous, where even hemotogenous infection may be a factor.

A single bacillus must frequently be deposited on the anterior part of the cartilaginous septum, the point where the inspired air first impinges, but, despite the frequent deposit of bacilli at this point and their proved ability to penetrate intact mucous membranes, infection is much rarer than in any other portion of the respiratory tract. Indeed, under normal conditions but few organisms pass into the nose and nasopharynx and from there into the deeper parts of the respiratory tract, for with its vibrissæ, mucus secretion and ciliary action the nose so perfectly performs its function as a filter that only an exceedingly small proportion of micro-organisms may penetrate beyond the introitus.

The endogenous infection occurs primarily in the course of an extensive pulmonary tuberculosis, usually as a terminal manifestation, and may be either hemotogenous or lymphogenous with extension to the mucous membrane from the underlying structures.

The hemotogenous conveyance of the organism has been demonstrated in the experiments of Mishulow and Park[18] in this country and of Theissing[30] abroad. The former research workers show that there is a rapid localization of tubercle bacilli when introduced into the blood stream of rabbits, as evidenced by the tremendous decrease in their number between twelve and twenty-four hours after inoculation, and by the continued decrease up to the fourth day. Although they fluctuated in number from day to day, the organisms persisted in the blood stream of the animals throughout the entire course of the infection. However, a marked rise in number of the bacilli in the blood was observed on the day of death. These investigators conclude that in the tuberculous there is a steady dissemination of tubercle bacilli into the blood stream and from there into the different tissues. In Theissing's[30] experimental work, pure cultures of tubercle bacilli were injected into the exposed common carotid arteries of rabbits and guinea pigs. The results of the histologic examination proved that the nasal mucous membranes and the bones of the nose were generally and simultaneously invaded. Though the areas of predilection in the nasal mucosa could not be marked definitely by the experiment, the nasal septum was very frequently and seriously in-

volved. In the experimental hemotogenous infection the bone destruction was brought about by a slow advance downward from the mucosa and also from the extremely numerous foci in the bone marrow. Penfold and Butler,[21] in their experiments, have also isolated tubercle bacilli in the blood of patients with tuberculosis.

The exact mode, or better the bioelectrophysics, as Mudd[20] terms it, of penetration or exclusion of tubercle bacilli from the mucous membranes is a complex one and, to a large extent, unsolved. Earlier workers thought that the organisms were engulfed by the epithelium or phagocytosed, but Maximow and Bloom[17] and Ross[26] contend that the epithelial cells in general do not display phagocytic activity. It was also conjectured that the entrance of the organisms into the tissue was caused by the ameboid movements of the epithelial cells. However, this activity is displayed usually only by the cells of the connective tissue. Though it is believed that epithelial cells may overtake organisms by a series of gliding movements, very little as yet is known of this mechanism.

That the ingestion or penetration of tubercle bacilli into the membrane is not purely one of absorption is shown by many research workers, such as Wilson,[34] Calkins,[3] Loeb,[15] Jennings[10] and Mudd.[20]

Wilson[34] supposed that the reaction between the protoplasm of the epithelial cells and that of the bacteria represented the same phenomenon as that of kinesis of cell division. He found such suggestion also in the gyrations of monocellular organisms around one another and around the tissue cells. This motion of either inanimate or living particles around tissue cells, he believed, represented adjustments to surface tension. Others explained the penetration of organisms into the tissue by a positive or negative chemotaxis or interaction between the bacterium and the surface of the epithelium. Under certain conditions, both general and local, the selective mechanism of the surface epithelium excludes some organisms and materials though it permits the penetration of others. This selective action is likewise attributable to the variation in the indices of surface tension between the bacteria and the cells. Wright[35] and other investigators have thought that the metabolism of proteid into fat and a fat absorption at the surface might account for the assimilation of bacilli by the epithelium because the fats and their derivatives, the lipoids, have been found in and about the tissue cells. In addition, it is to be recalled that the tubercle bacillus has a fatty or waxy capsule.

According to Wright,[35] living organisms by mere contact with mucous membrane can generate an electric current, and he thinks

that the lipoproteids are the main elements which take part in this electrochemical phenomenon. Therefore, in biochemical study the phenomenon of the invasion of the epithelium by the tubercle bacillus may be explained by the electrodynamics at the surface of the epithelial protoplasm, by the action of the colloids, by intracellular dynamics, and by the chemistry of the lipoids, etc.

Wright[35] says of Jacques Loeb:[15] "He has elucidated a biochemical mechanism in muscle cells, due to the currents of electricity set up, not by contact, but by induction in an exposed nerve, and this resulted in the formation of certain chemical changes in the terminal filaments of the nerves which, in turn, caused muscular contraction by virtue of the difference created in surface tension. Thus, the same process of modifying the surface tensions between the epithelial cell and the bacterium in the nasal mucous membranes by nerve excitations may determine the penetration or exclusion of the bacteria from the tissues."

From the foregoing statement it may be inferred that the pulmonary activity, or certain nasal pathology, may produce a reflex excitation which, in turn, influences the surface tension of the cell, thus inviting the penetration of the bacteria into the mucous membrane.

The penetration of the organisms, therefore, into the tissues, whether the result of phagocytosis of ameboid or gliding movements of the epithelial surface, or of some more direct biochemic change, is most satisfactorily explained by variations they bring about in surface tension.

That the tubercle bacillus is pleomorphic is an accepted fact. Under proper conditions bizarre forms, with lateral branchings or club-shaped ends, or even vacuolated types may be present and, in fact, are frequently found in the nasal secretions. These variation forms are not uusally found, though, when acutely progressive disease exists.

Spengler[27] and Much[19] have described fragments of tubercle bacilli as granular forms which appear to be resistant types of the organism and are found in those lesions where the rod forms are scarce. This scarcity results from the strong lytic action or unfavorable growth conditions existing in the nasal passages and probably accounts for the preponderance of granular forms found in the specimens used for this study. These fragments are avirulent or even nonvital, though they are capable of reproducing rod forms by inoculation or culture, as was evidenced in several of our inoculation experiments.

A study of the chemical pathology of the organism shows that a nucleic acid or tuberculinic acid is produced by the decomposition of the nucleoprotein substance present in the organism. This chemical substance Von Behring[31] considered the necrotizing substance in the bacillus that produced the caseating lesions, but many of the other local effects of the bacillus were produced by its waxy substance or its derivatives, the fatty acids.

In must be recalled that it was early demonstrated that dead bacilli could produce tuberculous lesions identical with those caused by live bacilli, but, as a rule, caseation was not present where the lesion was produced by dead bacilli unless a large amount of the organism had been introduced into the tissue. Since the nasal mucous membranes are so rapidly destructive to the tubercle bacillus, non-caseating tuberculous lesions are the rule in patients with primary nasal tuberculosis. In other words, the usual case of nasal tuberculosis may be produced by avirulent or even dead tubercle bacilli or their chemical products.

Thus, whether or not the invasion of the mucous membrane produces a local tuberculous lesion depends upon the virulence of the organisms and the numbers introduced into the tissues; upon the general resistance of the individual and his susceptibility to the organism, and upon the local nasal resistance provided by the action of the cilia and the protecting qualities of the mucous membranes and their secretions.

Previous methods of studying tubercle bacilli in the nasal mucous membrane were the following: swabbings and tissue removed by biopsy were studied microscopically and culturally; and in addition vital and supravital staining methods were applied in the investigation of the nasal mucosa. To the use of some of these tried methods I wish to add another—that of epithelial scraping.

Fifty unselected cases of active pulmonary phthisis in varying degrees of severity and that gave no clinical evidence of nasal tuberculosis formed the basis of the investigation. These were grouped according to the Turban-Gerhardt classification.* These cases were selected because they are the most likely to be exposed to nasal infection with the bacillus, and because they are proved to be tuberculosensitive.

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*The stage of pulmonary condition is given according to the Turban-Gerhardt classification of pulmonary tuberculosis: (Group I, slight involvement; group II, severe involvement; group III, advanced involvement.)

After the nasal vestibule was thoroughly cleaned with soap and water, the vibrissæ shaved, and the introitus painted with tincture of metaphen, a smear with a sterile swab was taken from the nose in every case. Slides were made and some of the material was put into sterile physiologic salt solution for inoculation purposes. The nose was then thoroughly irrigated with normal saline, all secretions and custs were removed and the membranes of the anterior septum, the turbinates and the floor of the nose were mopped dry with pledgets of sterile cotton. Next the mucous membranes of the anterior third of the inferior turbinates, the anterior septum and the floor of the nose were scraped. The instrument used in the process was a sharp hoe-like platinum spatula about five millimeters in width at the tip, flattened and slightly curved.

In obtaining the specimens the sharp edge of the spatula was placed back about the middle of the inferior turbinate well in contact with the mucous membrane, and with a quick outward movement some of the epithelium was scraped off. In like manner, specimens were obtained from the floor of the nose and the septum, great care being exercised at all times to avoid contamination from the vestibule.

The first scraping was put in sterile normal saline; subsequent scrapings were put on glass slides. These were stained according to the accepted method for demonstrating acid-fast organisms, and were examined microscopically. Guinea pig inoculations were made from the scrapings taken from the respective test tubes, but not without the realization that animal inoculation is not the final proof in such a case as this, since, as Havens notes, many will give negative results in positive cases. Though this method of animal inoculation, together with other methods of study, was included in the experimental work, most weight was given to the finding of the organism microscopically in the excised piece or in the scrapings of the tissue. The age and sex of the individual, any evidence of tuberculosis elsewhere in the body, and regional lymph node and clinical nasal findings were recorded. The tissue scrapings with their rod and variation forms, together with organisms other than tubercle bacilli were charted.

Analysis of the series of cases revealed the following facts: in every case, nasal epithelium was obtained on scraping; of the fifty unselected cases of pulmonary tuberculosis, fifteen, or 30 per cent, of the scrapings were positive for tubercle bacilli; of this percentage but one showed rod forms alone; seven, or 14 per cent, had both rod and variation forms of the organism with the former predominating, and seven revealed variation or granular forms only; hence, fourteen, or 28 per cent, of the positive series manifested variations or granular

forms either alone or in combination with rods. In all the cases where the nose was swabbed no organisms were found in the smears; yet in these same cases, by the method of epithelial scraping, tubercle bacilli were recovered in 30 per cent of the number. This fact forces one to the conclusion that the organisms had either penetrated the surface of the mucosa or that they were there by reason of hemotogenous invasion.

That tubercle bacilli may be obtained by swabbing was shown by the following investigators: Strauss[29] found bacilli in the nose of nine patients of the twenty-nine examined; Jones[11] found the tubercle organisms in 10 per cent of a group of normal individuals studied; Kistner[12] reported positive findings in a small percentage of normal noses; and Massione,[16] Cobb and Nagle,[4] and Wright[35] mention finding the tubercle organisms occasionally in normal nasal passages. But since we have no exact data relative to the experimental procedure involved, on a comparative basis these findings are not advisable or comparable to the findings obtained by scraping.

If the positive cases in this series may be considered as potentially tuberculous, the age and sex incidence is interesting, for, as Havens and other investigators have noted, the disease occurs in greatest proportion in the female, and in middle or late life in either sex. All of Haven's[9] five patients with primary nasal tuberculosis were women past fifty years of age. Raudhitz,[23] Pontoppidan,[22] and Bloch[2] in three different series of nasal tuberculosis reported 34 per cent males and 65 per cent females. Of Freer's[6] two patients, one was a young woman of twenty and the other a woman of fifty. Warde's[32] patient was a woman of thirty-nine and Kistner's a woman of seventy-three.

In contradistinction to these findings relative to age and sex, thirty-seven was the average age of the fifty unselected patients examined as a basis for this study, and thirty-two was the average age of those affording positive findings. The youngest subject was sixteen years of age; the eldest was sixty-one. In our positive cases, 60 per cent were males and only 37 per cent were females.

Of the clinical findings, the most striking were those of the septum. Deviations of the nasal septum were present in thirty-nine, or 78 per cent, of all the cases examined. Of the fifteen cases having tubercle bacilli in the nasal mucosa thirteen, or 86 per cent, had pathologic deviations. The majority of the deviations involved the bone and cartilaginous parts of the septum, two of which had bony spurs. In the two cases of the series that had had previous submucous opera-

tion, most of the deflection was uncorrected and perforations in the cartilage were evident. In three cases of the series, superficial crust-covered, nose-pickers type ulcers of the anterior septum were found. Biopsy and tissue scrapings from all three of these cases yielded no evidence of tuberculosis, nor were any tubercle bacilli found. Many investigators find septal ulcers more frequent in the tuberculous than in the normal individual. Kyle[13] found simple septal ulcers in one case out of every twenty-five patients treated for pulmonary tuberculosis; Weichselbaum[23] and Lockard[14] claim that simple septal perforations are found twice as often in the tuberculous as in the normal patient, and contend that many of these ulcers, in fact, depend on tuberculosis.

Atrophy of the nasal mucous membranes was present in varying degrees in thirty, or 60 per cent, of the series examined, and hypertrophy of the turbinates was noted in three, or 6 per cent. The greatest atrophy was found on the side of the greatest concavity of the deflected septum. Freudenthal,[7] Hamilton[8] and Alexander[1] found, however, an average of 25 per cent of nasal atrophy in a varying series of patients with pulmonary tuberculosis. Thirty-four, or 68 per cent, of all our cases examined manifested pallor of the membranes of the septum and turbinates, and in seven, or 14 per cent, the membranes were hyperemic.

Other complications were noted in this study. Crusting was present in moderate amounts; in all, it was discovered in only five of the cases of the series and in only two of the positive group. There was no manifestation of ozena in our series. Polyps arising from the middle meatus were manifested in only one case, and here no tubercle bacilli were discovered by biopsy or scraping. This patient had asthma. Secretion, postnasal and from the middle meati, was present in two, or 4 per cent, of the cases together with other clinical evidences of sinusitis.

The incidence of clinical nasopharyngeal findings in percentage were as follows: Eighteen, or 36 per cent, of the series manifested moderate pallor of the pharynx and larynx, and sixteen, or 32 per cent, showed a general hyperemia of these parts. Of the fifteen cases yielding tubercle bacilli from the nasal membranes eight, or 54 per cent, exhibited hyperemia of the pharynx and larynx, and three, or 20 per cent, had pallor of the pharyngolaryngeal membranes.

In the fifty cases examined, seven, or 14 per cent, had tuberculous laryngitis in varying degrees of activity; and in those cases yielding tubercle bacilli from the nasal mucosa, but two, or 13 per

cent showed clinical tuberculous laryngitis. These percentages compare favorably with those found recorded in the scant literature on the subject wherein it is noted that about 10 or 12 per cent of persons with nasal tuberculosis have tuberculous laryngitis also.

Since this study deals but incidentally with nasal tuberculosis, these statistics cannot be considered directly comparable, but they are of interest. Regional lymph nodes were palpable in twenty-four of fifty cases, approximately 50 per cent. The submaxillary group were palpable in seven, or 46 per cent, of the fifteen cases yielding tubercle bacilli in the scrapings. Cough was present in 90 per cent of the whole series, and in fourteen, or 92 per cent, of the cases showing tubercle bacilli. Sputum analysis was positive in 82 per cent of all of the cases, and in the fourteen positive cases thirteen, or 86 per cent, had positive sputums.

Since the primary purpose of this study is to demonstrate the tubercle bacilli microscopically in the nasal scrapings, only from those cases yielding organisms on scrapings were guinea pig inoculation studies made. Of the fifteen positive cases showing rod and variation forms eleven, or 73 per cent, gave positive guinea pig inoculations. Of the eight cases yielding rod forms predominantly in the scrapings, all revealed tuberculous lesions in the peritoneum, liver and the lungs. Of the seven cases with variation forms only three developed tuberculous lesions of the liver and peritoneum and in this group rod forms of the organism were recovered from the guinea pigs. This proves the lesser virulence of the variation forms. In four of the total positive series tubercle bacilli were recovered from the blood stream. Four of the guinea pigs inoculated revealed no tuberculous lesions.

Many other phases of this study, such as nasal sinus findings, the cytology of the nasal secretions, and other clinical and laboratory analyses or data are mentioned; however, since these are outside the province of this experimental work they are noted but briefly in passing.

In order to clarify the findings, the following charts are prepared and are self-explanatory:

Chart I shows the incidence in percentage of the tubercle bacilli in the nasal scrapings together with the other microscopic findings;

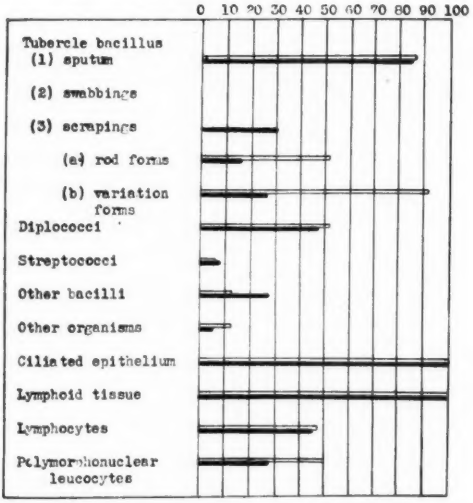
Chart II shows the incidence in percentage of clinical nasopharyngeal findings;

Chart III shows the frequency in percentage of the extent and character of pulmonary findings together with complications;

Chart IV shows results of guinea pig inoculations.

CHART I

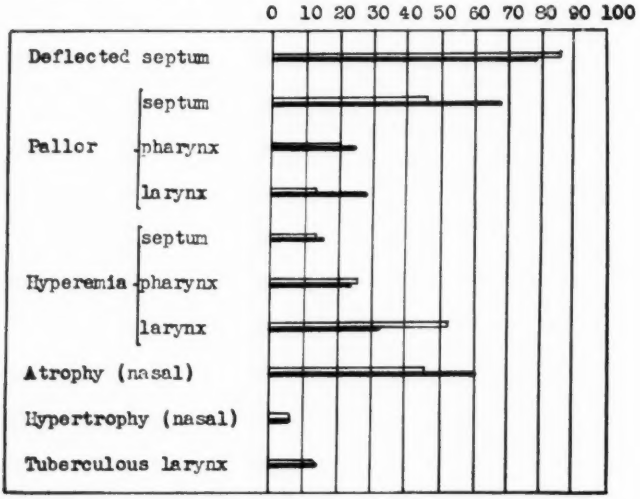
The incidence in percentage of the tubercle bacillus in the nasal scrapings together with other microscopic findings.



Open bars indicate percentage in patients having positive scrapings.
Closed bars indicate percentage in total number of patients examined.

CHART II

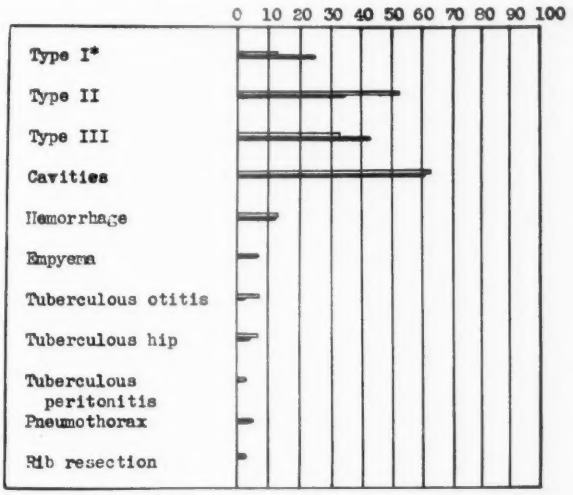
The incidence in percentage of clinical nasopharyngeal findings.



Open bars indicate percentage in patients having positive scrapings.
Closed bars indicate percentage in total number of patients examined.

CHART III

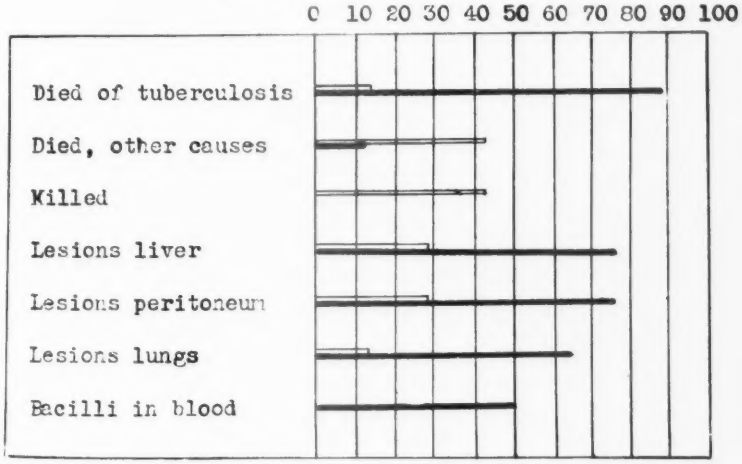
The frequency in percentage of the extent and character of pulmonary findings together with complications.



Open bars indicate percentage in patients having positive scrapings. Closed bars indicate percentage in total number of patients examined.

CHART IV

Results of guinea pig inoculations.



Open bars indicate percentage of pigs inoculated with variation forms. Closed bars indicate percentage of pigs inoculated with scrapings wherein rod forms predominated.

CONCLUSIONS

1. This method of epithelial scraping offers certain advantages in the study of nasal bacteriology in general, and is of particular value in showing the existence and incidence of tubercle bacilli in the mucosa of the tuberculous.

2. Of the fifty unselected cases of pulmonary tuberculosis, 30 per cent showed tubercle bacilli in the epithelial scrapings, but in the nasal smear of these same cases no organisms were found.

3. Eighty-six per cent of the cases showing tubercle bacilli in the nasal mucosa had pathologic deviation of the nasal septum.

4. Age and sex incidence was inconclusive.

5. Entrance of tubercle bacilli into the nasal mucous membrane may be an ectogenous invasion caused by fingers, handkerchief or bacilliferous sputum, or endogenous by way of the blood stream and lymphatics.

6. Patients showing positive tubercle bacilli in the nasal mucosa may possibly be considered as potential cases of nasal tuberculosis.

7. Since 28 per cent of the positive cases yielded variation or granular forms of the organisms but only 16 per cent yielded rod forms, and since the variation forms are avirulent or dead organisms, and since the latter forms are found where the lytic action or the resistance toward the organism is great, one may possibly conclude that many cases of primary nasal tuberculosis are caused by avirulent or dead tubercle bacilli or their chemical products.

8. This method of epithelial scraping may be of value in studying the nasal bacteriology in different acute and chronic infections of the mucosa. Since the bacterial flora of the nose is so complex, and since it is difficult to determine which organism is the true infective agent, it is correct to conclude that the organism or organisms found subepithelially are probably the predominating organisms in the disease, and more than likely are the causative factors.

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XI

TEMPOROMANDIBULAR JOINT MALOCCLUSION AND THE INNER EAR, A NEUROMUSCULAR EXPLANATION*

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This paper presents certain impressions, gained from a preliminary study of malocclusion of the temporomandibular joint, which calls for otolaryngologic consideration and about which there appears but little reference in the otolaryngologic literature. Costen,[1] of St. Louis, has made notable contributions, however, within the past few years.

In the dental literature there is much more evidence of interest beginning with Wright of Pittsburgh, in 1920, and culminating in a contribution by Goodfriend[2] of Philadelphia, in 1933. Wright,[3] of Boston, called my attention to the subject in 1919, and refers to it in a paper before this Society in 1925.

Today the dental profession is earnestly seeking the co-operation of the otolaryngologists in the successful handling of cases of malocclusion. It is a problem calling for physiologic restoration of the normal mandibular posture, the loss of which creates a functional problem for the dentist, and the effects of which defy the otolaryngologist in that it disturbs the functions of hearing and speech, as well as mastication and deglutition.

Furthermore, it may seriously determine food selection, digestion, assimilation and elimination, with proportional degrees of malnutrition and systemic disease.

Food selection in these cases tends toward a highly refined carbohydrate, low mineral and low vitamin intake, inviting an imbalance in the usable calcium-phosphorus ratio, with increased disturbance of dental metabolic equilibrium. One is impressed by the subtle progress, with short irritative phases and longer quiescent intervals of

*Read before the Eastern Section meeting of the American Laryngological, Rhinological and Otological Society, Boston, January 7, 1937.

readjustment, such that they escape recognition of the true significance of future trouble.

This fact is well illustrated by a recent conversation with one of the outstanding otologists of this country. When asked how many cases of this kind he had observed in his practice, he replied that he had never seen any that he could recall.

The temporomandibular joint occupies an important key position, due to its commanding location in nutrition, and its intimate association and influence in the actions and reactions of the 5th, 7th, 8th, 9th and 10th cranial nerves and their higher centers of control.

By the time an acquired malocclusion is recognized there usually is at least a minor vicious cycle in progress. For example, in those cases where the acquired malocclusion starts in the first decade of life, with the loss of a six-year molar, there probably is a pre-existing nutritional defect, starting even during the critical period of prenatal life, in which a mineral and vitamin deficiency existed, plus any hereditary endocrine tendencies.

Such a background is setting the scene for a gradual onset of a condition that may slowly, even clandestinely, progress during the first three or four decades, before the major manifestations appear. After forty, the recuperative powers are not as great, the effect of infections have made their inroads, the economic responsibilities of life have usually reached their peak and the weaker links begin to let go, through degenerative changes. So it is that the majority of cases are appreciated in the later decades of life.

SYMPTOM-COMPLEX

The symptom-complex associated with malocclusion may have a wide range, and will vary in direct proportion with the individual background upon which it has been superimposed.

There may be appreciable changes in the tonicity of the musculature of mastication, facial expression and general skeletal posture.

There may be reflex pain to one or both sides of the head, often called neuralgic headache by the patient, radiating to the face and neck; vague discomforts referred to the eye, ear, nose or throat, particularly in the hyoid region; dryness of the mouth due to inadequate salivation, accompanied by dysfunction, swelling and induration of the salivary glands, inviting incomplete mastication and frequent drinking of fluids to wash down the food; paresthesia of the pharynx and at the base or side of the tongue, frequently interpreted by the patient as indicative of cancer of the throat; otalgia, itching of the

external canals, herpes, stuffiness of the middle ear and the symptoms referable to the inner ear, namely, tinnitus, deafness and vertigo.

There may be functional disturbances of the general nervous system, through fear and pain, such that personality changes develop, even to neuroses and psychoneuroses.

DIAGNOSIS AND PROGNOSIS

The diagnosis is simple enough. The principal difficulty is to include it in the differential diagnosis and to evaluate to what extent it is a factor.

Abnormalities in the joint, such as excessive excursion of the head of the condyle, crepitus and tenderness may be detected by palpation, while lateral excursion of the mandible in opening and closing may be observed by inspection. When dentures are present, their removal may give added information as to their adequacy.

Finally, corroboration of whether opening the bite will relieve the symptoms may be determined by having the patient wear dental cork disks two millimeters thick, according to Costen.[1] The trial dental splint, described by Greene, would appeal to me as more satisfactory.

The prognosis depends upon the cooperation between the patient, the dentist and the otolaryngologist.

STUDY LIMITED TO MALOCCLUSION AND INNER EAR. OFFERING AN EXPLANATION

This study centers about two clinical facts:

1. That malocclusion of the temporomandibular joint may be associated with a wide range symptom-complex.

2. That the symptom-complex may disappear in proportion to the successful restoration of the physiologic function.

This paper principally concerns itself with the explanation of the connection between the malocclusion and its effect upon the inner ear, namely, tinnitus, deafness and vertigo.

An explanation will be offered to show that the connection between the disturbance of function of mastication and the disturbance of function of the inner ear is by virtue of a disturbance of function of the tympanic and tubopalatal muscles.

Let us now consider the origin and role of certain ones of these muscles which play the important part.

THE MUSCLES OF MASTICATION, DEITER'S NUCLEUS AND VERTIGO

The musculature of mastication, together with the mandible, develops from the first visceral or branchial arch and is innervated by the fifth cranial nerve-mandibular division.

If it is true, as Lord[4] maintains, that the mandible is closed and held closed by the temporal, masseter and internal pterygoid muscles, then they can be proved to be antigravity or extensor muscles by experiments on decerebrate animals (Cannon[5]). This is important to keep in mind in considering postural tonus of these muscles under normal and abnormal conditions. Under normal conditions these posturing muscles are relatively indefatigable (Cannon[5]).

Malocclusion is the result of the lack of the normal molar control in the determination of the normal amount of closure of the bite and the position of the condyle in the glenoid cavity. The constantly shifting relationship of the attachments of these muscles to the mandible may create loss of tone in some portions and increased tone in other portions of the same muscle.[6] This may occur more in the temporal than in the other two muscles. With the shifting of the axial planes of the remaining teeth, following the loss of the normal support, there is a constantly increasing vicious cycle at work in which an ever increasing tactile sensation, amounting to painful stimulation, is taking place around the necks and apices of the shifting teeth with pyorrhea and apical infection as secondary factors.

The tone in the anti-gravity group of muscles of the mandible represents a state of reflex contraction which is concerned with maintaining position or posture, and as posture is the basis of movement, all mandibular movements begin and end in a posture.

It is maintained that in malocclusion there is abnormal position or posture and therefore abnormal movements begin and end in abnormal posture; that the abnormal posture and movements during mastication initiate abnormal afferent (proprioceptive) stimuli which create, through reflex action, an increased muscle tone with abnormal pressure either in the joint or on the axial planes, particularly of the incisor teeth, creating additional afferent (exteroceptive) stimuli within the distribution of the fifth cranial nerve; and that the tensor tympani muscle shares similar changes in muscle tone, with the antigravity group, such that there is actuated an increased intralabyrinthine pressure, transmitted through the ossicular chain and footplate of the stapes.

Changes in intralabyrinthine pressure are capable of exciting the end organ of the vestibular nerve, with resulting increased stimuli

(proprioceptive) passing from it to Deiter's nucleus. As Deiter's nucleus has an important influence in maintaining tonus normally, and protects the "final common path" from inhibitory stimuli (Magnus[8]) it would be reasonable to expect, under abnormal stimulation, that it would actuate an abnormal increase of tonus.

It may be seen from these conditions that a vicious cycle may at times be initiated to a clinical degree, in the acute irritative phase of a malocclusion, capable of producing the pathologic symptom—vertigo.

It is conceivable that Deiter's nucleus under prolonged repeated over-stimulation may become an "irritable focus" similar in principle to that of the posterior horn cells described by MacKenzie[7] in heart disease, namely, an "irritable focus" or "hypersensitivity" to stimuli that under normal conditions would fail to be adequate stimuli.

Magnus[8] considers Deiter's nucleus the principal brain stem center for proprioceptive stimuli from the vestibular nerve, peripheral distribution, while proprioceptive stimuli from the muscles, tendons, ligaments, joints, periosteum and bones, according to Davis,[9] relay through a simple reflex arc in the brain stem.

Magnus[8] also considers the reflex mechanism necessary for the maintenance of postural tonus to be conditioned by one important factor, namely, the tension exerted upon the anti-gravity muscles in question by their normal anatomic attachments. Furthermore, Magnus[8] considers the presence of the vestibular nuclei necessary for the continuance of well marked myotatic reflexes, and that this center must exert a constant influence on the local inhibitory pathways such that the neurones of the "final common path" are relatively inaccessible to inhibitory impulses.

In the case of the temporomandibular joint it is reasoned that the mandible is maintained in posture by the antigravity muscles (temporal, masseter and internal pterygoid) and that in malocclusion there is clinical evidence, on occasions, of increased and decreased muscle tonus. The increased muscle tonus is explained by increased tension, exerted upon the antigravity muscles, by their now abnormal anatomic attachments, both while masticating and seeking posture. It is reasoned that increased functional and postural tonus is exerting a molding force on the axial planes of the remaining teeth and that this undue pressure gives rise to exteroceptive stimuli in the dental distribution of the mandibular and maxillary nerves (third and second divisions of the fifth nerve respectively) and that the stimulation of the sensory portion of the fifth nerve is not only

capable of causing reflex pains throughout its distribution, but it is capable of producing vertigo, by virtue of variations of changes in the muscle tension of the tensor tympani muscle.

THE ORIGIN AND ROLE OF THE TENSOR TYMPANI MUSCLE

The tensor tympani muscle has the same visceral, embryonic origin as the muscles of mastication, namely, the first branchial arch, and they are all motivated by the same nerve—the fifth cranial—mandibular division. It is important to bear in mind that stimulation of the sensory division of the fifth nerve (Rioch[10]), and particularly the mandibular division, evokes powerful reflxes involving all these muscles, including the tensor tympani.

When the tension of the tensor tympani muscle is altered from the normal posturing tone, the conditions of the inner ear are changed so that the functions of the organ of Corti and the utricle are altered.

Changes in the cochlear reflex, affecting hearing, are now under intensive research study by Davis, Lurie and co-workers, at the Harvard Medical School. By means of animal experimentation, using electrical stimulation of the cochlea, they are able to detect the results, by the cathode-ray oscillograph, in which auditory stimuli are producing reflex stimulation of the tensor tympani muscle. Lorente de No[11] cites evidence that not only faradic stimulation of the afferent pathways of the fifth, but those of the auricular branch of the tenth, causes a reflex stimulation of the tensor tympani and stapedius muscles.

It is assumed that in malocclusion there is sufficient evidence of painful stimulation, in the sensory distribution of the fifth nerve, especially the mandibular division, to produce reflex contractions of the tensor tympani mucle, so as to create muscle fiber contractile sounds, which would account for not only the tinnitus but the diminution of the hearing—a transmission deafness (Lurie[12]).

Furthermore, the changes in tension in the tensor tympani may, at times, be increased to such an extent that it is capable of becoming an adequate stimulus to the vestibular nerve end organ (utricle) such as to produce the dizziness—of external origin.

It is assumed that other etiologic factors producing affections of the inner ear and the eighth nerve may have rendered important changes therein, so that the superimposed effect of malocclusion is but an additional, external factor. The acute exanthemata of childhood, grippe, chronic upper respiratory infections, the toxins of focal infection, renal toxemia, the toxic action of tobacco, alcohol, quinine

and the salicylates, anemia, changes in blood pressure, arteriosclerosis, lues, endocrine imbalances, fear, are all factors capable of causing such changes.

Disturbance of the normal aeration of the middle ear is attributable to the disturbance of function of the tubopalatal muscles, notably, the tensor palati.

THE ROLE PLAYED BY THE TENSOR PALATI MUSCLE

Of the muscles of the eustachian tube regulating the aeration of the middle ear, probably the most important one is the tensor palati. It receives its motivation from the fifth cranial nerve, and so may be considered to have its visceral, embryonic origin in common with the tensor tympani and masticator group of muscles.

It is therefore assumed that, like the tensor tympani, the tensor palati will be affected by painful stimulation of the sensory distribution of the fifth nerve, especially the mandibular division. As "stuffiness" of the middle ear is a common complaint in malocclusion, it is assumed that the function of the tensor palati, in opening the eustachian tube, may be inhibited. On the other hand, it may be thrown into a spasmodic contraction. Several authors, including Politzer[13] and Lüscher,[13] working independently, came to the conclusion that cases of objective tinnitus (audible clicks) were to be explained by the disturbed action of the tubopalatal muscles, especially the spasmodic contraction of the tensor palati.

Thirty-two years ago Wells[13] reported two cases of objective aural tinnitus due to the action of the tubopalatal muscles, and, in the case history of one, the patient volunteered the information that "when he opens his mouth wide he has a sense of grinding at the point of articulation of the lower jaw."

Weil[14] states that the clicking sounds in objective tinnitus, like the snapping of two finger nails together, is caused by a clonic spasm of the muscles in and around the eustachian tube and that the muscle most commonly affected is the tensor palati.

The other two tubopalatal muscles (levator palati and salpingopharyngeus) take their visceral, embryonic origin, along with the other palatal, pharyngeal and largyngeal musculature, from the third and fourth branchial arches and are innervated by the ninth, tenth and accessory portion of the eleventh cranial nerves.

In Wells'[13] case reports of objective aural tinnitus, he states that "on inspection of the throat it was seen that the muscles of the

pharynx and palate were involved in an almost continuous clonic spasm, which accompanied the up-and-down movement of the larynx and was isochronous with the clicking noise in the ears." This occurred in the same case referred to above as having a malocclusion of the temporomandibular joint.

From these facts it is assumed that the malocclusion of the temporomandibular joint disturbs the normal functions of the tubopalatal musculature, in aeration of the middle ear and in deglutition, by disturbing the voluntary portion of the musculature of the palate, pharynx and larynx.

In other words, a mechanical disturbance of the mandible and its joint causes a mechanical disturbance to the middle ear and inner ear, producing the symptoms of tinnitus, deafness and vertigo.

The vertigo is the result of peripheral irritation, from changes in the tension of the tensor tympani muscle, plus the changes in the middle ear, from inadequate aeration, due to disturbances of the function of the tubopalatal muscles, especially the tensor palati.

ROLE OF THE STAPEDIUS MUSCLE

The role played by the stapedius muscle should be considered next. It has the same visceral, embryonic origin as the muscles of expression and, like them, is motivated by the seventh nerve. However, it differs from the facial group in one very important respect, namely, although a skeletal muscle, it is not under voluntary control but under involuntary reflex control.

According to Lüscher,[15] of the University of Berne, the stapedius muscle plays a role in the middle ear, not so much to aid hearing as to protect the organ of Corti, by a "damping effect." He studied the stapedius muscle through an unirritated, large, posterosuperior perforation of the eardrum, in an otherwise normal middle ear, by the use of an ear microscope (50 magnifications). He demonstrated that not only acoustic stimuli but painful stimuli are capable of stimulating contraction in the stapedius muscle, by means of a simple reflex bulbar arc; that the stapedial reflex is very sensitive to even slight stimulation—such as the soft noise of the breath at the ear or pricking the external ear, which will release the reflex contraction; that no movement of the drum occurs at the time of the contraction; and that the strongest voluntary contractions of the muscles of expression are not accompanied by a contraction of the stapedius, e. g., a voluntary contraction of the stapedius cannot be established.

Lüscher[15] concluded that contraction of the stapedius muscle causes a decrease of the sound transmission and thereby a decrease in hearing.

Kobrach[16] observed that the stapedius muscle has a more sensitive threshold than that for the tensor tympani, and the threshold for hearing is more sensitive than the threshold for muscle contraction.

It is assumed that, in malocclusion, painful stimuli arising in the sensory portion of the third division of the fifth cranial nerve, furnish adequate stimuli to release the stapedial reflex, by way of the secondary sensory collaterals of the fifth nerve, in the reticular area of the medulla and pons, which have been demonstrated to have direct synaptic connections with the cells of the motor nucleus of the seventh nerve (Cajal[17]).

SUMMARY

The problem of malocclusion of the temporomandibular joint is one requiring both the dentist and the otolaryngologist. "Malocclusion consciousness" on the part of the otolaryngologist will automatically include this factor, in the differential diagnosis of causes of reflex pain in the side of the head, suggesting eye, ear and sinus disease.

The progress of the malocclusion is insidious over a relatively long period, and is marked by shorter irritative phases and longer latent intervals of temporary occlusal balance or posture.

The symptom complex is both local, neighborhood and systemic.

Its major phases are most noticeable in the later decades of life.

To paraphrase Evans and MacDonough's syllogism on hypoglycemia—"malocclusion consciousness" takes another symptom complex from the waste-basket of the neuroses.

CONCLUSIONS

1. The explanation of the connection between the malocclusion of the temporomandibular joint and the inner ear symptom complex is based upon a mechanical stimulation involving a neuromuscular set-up in the mandibular joint, reflexly involving the related neuromuscular set-up in the middle ear, with a resultant mechanical disturbance in the inner ear, signifying a biologic protective reaction to a harmful stimulus.

2. That this external peripheral stimulus will vary in its effects according to the stage of irritation; that tinnitus is of muscular ori-

gin; that the deafness is of the transmission type, and the vertigo is due to peripheral stimulation of the vestibular end organ, due to changes in the tension of the tympanic muscles, notably, the tensor tympani.

3. That the symptom-complex will be associated with other local and systemic factors and the whole picture will vary with the individual behavior.

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XII

TEMPOROMANDIBULAR JOINT: DENTAL ASPECT*

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Normal and abnormal movements of the temporomandibular joint have never received the attention they should, considering their importance physiologically and pathologically to tissues elsewhere in the body.

The intimate relationship of this joint to several cranial nerves, together with its complicated mechanism, makes the study of this region vital.

From early life when the mandible begins to emerge from under the skull to influence the shape of the face, the temporomandibular joint is normally concerned in such functions as mastication, deglutition, speech, middle ear ventilation, and facial expression; and it is also involved in systemic reflex actions.

From a dental standpoint only orthodontists as a group have stressed the importance of normal function in the mouth. However, the prevalence of malfunction and the time and expense necessary for its correction are so great that the general care of these cases is not possible.

The fact that young people compensate so readily to malfunction in this region makes dentists themselves overlook the abnormal reactions that are developing from this condition. Thus, many of them fail also to connect the serious systemic disturbances that occur in later life with this childhood loss of the normal posture of the mandible.

The public's interest in dentistry centers around the relief of pain and the replacement of missing teeth, the latter mainly for esthetic purposes.

People must be taught that normal function in the oral cavity is a great aid in maintaining general health and that occlusion is the vital factor.

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Let us consider what normal occlusion is. I like Le Roy Johnson's definition:[1] "Normal occlusion is a condition of occlusion that is functionally in harmony with the metabolic processes that maintain the supporting and the surrounding structures of the teeth and jaws in a state of health."

Malocclusion occurs when the first permanent molar is either lost, malposed, or through caries, loses any of its articulating surfaces. Any change from normal, either in form or position, creates disturbance in the temporomandibular joint through loss of masticating equilibrium. Thus the welfare of the joint is dependent upon the welfare of the teeth.

In the mouth there are gradual changes in the alveolar processes which in later life, when the general resistance to disease is lowered, are recognized under the general term of pyorrhea.

There are changes in the shape of the mandible at the angle, the neck and the head; also changes in the articulating cartilage, and in the glenoid fossa. There is a stretching of the ligaments, a loss of tone and co-ordination in adjacent muscles, an interference with nerve functions, and altered dietary habits from lowered masticating efficiency. These results demonstrate the physiologic law that form depends upon function.

These changes as they relate to reflex symptoms in other parts of the body have never been classified. Here is a field that needs investigation.

Goodfriend[2] of Philadelphia has described the changes which affect the temporomandibular joint as follows:

> "A partial dislocation is called a subarthrosis. This is the forerunner of all mandibular disarticulations of a chronic or recurrent type. It has been called subluxation. Its local symptoms are crackling noises, lateral displacement of the meniscus, partial occlusion and trauma of the external auditory canal with painful and spastic movements of the joint. The crackling noises are probably caused by a loosening of the attachment of the disc to the condyle. Snapping of the condyle is probably due to the retardation of the gliding movements in the upper joint and is caused by the contact of the roughened upper surface of the meniscus with the glenoid fossa."

The complete dislocation he calls a dysarthrosis. It can be either forward or backward and either bilateral or unilateral. In a forward bilateral case both mandibular condyles completely slip over the eminentiæ articulares. Posterior dislocation is a condition in which the condyle leaves the glenoid fossa and articulates upon the greater wing of the sphenoid bone. It is comparatively rare.

It requires no scientist, however, to recognize that disturbed relations of the human jaws are prevalent. Anyone who observes the jerky masticating movements that are made by many patrons of restaurants can appreciate that the joint is under abnormal tension and that efficiency is below par.

As this pathology in the joint relates directly to the ear, I shall quote what Crane, a physician, wrote in the New York Dental Recorder in 1854:[3]

HEARING RESTORED BY ARTIFICIAL TEETH

"I am confident that some readers will feel disposed to ridicule and disbelieve in any such means of restoring hearing, but they cannot object to a statement or facts which have occurred of late. Miss T. has been partially deaf ever since I have known her, but how early the trouble commenced or what was its cause I have no knowledge. She began to lose her teeth early, when dentistry was in its infancy, and those that were not destroyed by caries became loose, until all had disappeared. Her gums were soft and spongy before the teeth were removed, but healed readily after that operation.

"She then had a full set of artificial teeth inserted, which gave her constant pain and trouble for three years, and occasioned greater absorption on the lower jaw than I ever witnessed in any living being.

"Her dentist persuaded her that all this was to be expected, but it would all come right in time, and induced her to persevere; the absorption continued until the under jaw was little more than half an inch in depth. The teeth he made for her were much too short.

"During the three years' martyrdom, the masseter and buccinator muscles became contracted, and her face consequently much shortened. In this condition she applied to me for relief.

"The usual means were employed for restoring the symmetry of the face, and the teeth were necessarily made much longer than those she had previously worn. The first few days after they were inserted, she complained of pain in the muscles of the jaw, and on examination I found them to be inflamed and considerably enlarged; the parotid gland was also swollen and painful; the gums being but slightly inflamed. I concluded that the stretching of the muscles was the cause of the inflammation and enlargement of the parts, and an application of the tincture of arnica soon returned them to a healthy state.

"In about two weeks the lady informed me that her hearing had improved: a result as unexpected as it was agreeable. She could hear better than she had done for years. Of course, this gave her much joy, and I truly hope and trust that it will prove to be a lasting comfort and blessing."

Other symptoms, such as vertigo, tinnitus and neuralgia in adjacent tissues, react to the repositioning of the mandible, and many cases are recorded in which relief has been obtained by this treatment.

Several different theories have been advanced to explain how the disturbed joint relation causes these ear syndromes.

One is that encroachment upon the external auditory meatus caused by the intrusion of the head of the mandible directly interferes with hearing. Wright of Pittsburgh[4] expressed this view in Boston at the National Dental Meeting in 1920.

Another theory is that dysfunction in mastication and deglutition prevents proper ventilation of the middle ear through constriction in the eustachian tubes caused by the impaired action of adjacent muscles. Costen brought out this theory in 1933:[5] The causes of the neuralgias, he thinks, is the impaction of the auriculotemporal and the chorda-tympani nerves, due to the uncontrolled movements of the mandible.

However, the improvement that occurs in the general health, mental attitude and expression, as well as in the adjacent tissues, is so marked in patients who have been treated for this joint disturbance that a more vital factor must be involved than that created by changes in form of either bone or muscle tissues.

The fact that in full systemic muscle function the normal joint is subjected to a severe balanced tension, suggests this theory to the writer: When this tension is lost through malocclusion and disease, the proprioceptor nerves create impulses that transcend the threshold of normal nerve function. These impulses then follow a special pathway to communicate with the central nervous system. The reflexes resulting from this change in transmission affect unfavorably other body tissues.[6]

The dental treatment necessary to correct pathology in this joint is to reposition the mandible to its normal relation with the glenoid fossa. Because of the changes that have occurred in both the hard and soft tissues in this region, considerable dental skill is required to reëstablish proper relationship and to restore function satisfactorily through the use of any type of mechanical restorations. To minimize the risk of error and unnecessary expense it is better to use temporary adjustable bite splints until relief is assured. Before describing these interdental splints, I would like to classify the types of mouths most likely to cause reflex disturbances.

First, those in which there has been lost a great amount of tooth surface from excessive wear, but in which teeth have remained firmly held in their sockets.

Second, those which through attrition, caries or extractions have lost normal tooth contact in the molar regions.

Third, those in which restorations have been made which have not restored masticating equilibrium.

Fourth, those which are edentulous.

These types create a gradual, but a continual loss of the vertical distance between the jaws, the biting force is no longer exerted on the long axes of the teeth, the joint becomes traumatized, and this may result in the perforation of the cartilage.[7] In most cases the underlying bone is more seriously affected than the meniscus. The loss of space shortens the distance between the origin and insertion of the extensor masticating muscles, and conversely it stretches the fibers of the flexor muscle group. The resultant change in the tone of these muscles interferes with the mandible's maintaining its resting posture.

A practical way to feel the reactions that occur when there is much disharmony in the joint is to stand in front of the patient, put both thumbs on the forehead and the little fingers in the ear canals. The condyle heads of the mandible can be located when the patient opens and closes the mouth. The backward thrust of the mandible will be felt when the excessive closure takes place. A temporary stop between the incisor teeth, such as a wooden tongue depressor, will prevent this backward thrust on closure, if it is of the right thickness to maintain the normal space.[8]

Splints are usually adapted to the biting surfaces of either the natural or the artificial teeth of the lower jaw, and are attached to them with cement. If no teeth are present, fitted blocks are employed. A trough on the superior or working side of the splint is filled with cement to establish a working occlusion. The distance between the jaws can be altered by increasing or decreasing the thickness of the cement. This adjustability enables the patient to adapt himself to changes, and to help the dentist establish the proper joint relationship and also to watch the soft tissue reaction before the details of the permanent restorations are planned.

Radiographs of the joint have real diagnostic value. Risner advocates repositioning the mandible by taking a series of x-rays of the joint with the distance between the jaws changing two mm. with each picture until the heads of the condyles are shown to be normally placed just behind the eminentiæ.[9]

In long standing edentulous mouths the shrinkage in the masticating muscles is so great, and the tongue has become so flattened that to restore full vertical correction with one set of dentures makes the new teeth so conspicuous, and the cases so clumsy, that it is not economically sound in the average dental practice to even attempt this correction. The result is that the patients with serious joint disturb-

ances seldom obtain the appearance, efficiency and comfort that they should receive when restorations are constructed. Some cases, to get proper results, must be treated through a series of appliances.

Otolaryngologists could greatly assist dentists in controlling these patients, by advocating that dentistry for function and not for esthetics is the type of treatment that they require.

When the mandible is repositioned through the occlusion of new balanced dentures, masticating efficiency can be improved more rapidly by utilizing myofunctional therapy as developed by Alfred P. Rogers in orthodontic treatment.[10] The muscles used in speech will also respond to well planned exercises and hasten improvement in articulation.

I should like now to present two brief histories of patients who have had joint disturbances.

REPORT OF CASES

CASE 1.—A boy, 5½ years old, was referred by Dr. Seaver, an otolaryngologist, because of a tongue-tie and a speech defect. He was badly undernourished, weighing only thirty-one pounds. Four different physicians had been unable to help him. There was such a wide overclosure of the bite that his lower teeth closed inside his upper teeth until they met resistance in the mucous membrane of the upper jaw. This situation interfered with his jaw joint movements, and the closed bite prevented the tongue from adjusting itself satisfactorily in the small oral cavity. This created a drainage problem in the throat and inhibited the normal development in the positioning of the hyoid bone to the larynx. He presented a picture often described as that of an endocrine disturbance.

Orthodontic treatment by Dr. C. P. Bonin was started about July 15th, and on December 1st the boy weighed fifty pounds. His appearance, posture and other systemic reactions were equally improved and Dr. Seaver reported sufficient progress in speech to make a tongue operation unnecessary.

CASE 2.—A woman, 72 years old, had worn full dentures for seventeen years with the usual shrinkage of the ridges taking place and overclosing the bite, which shifted the chewing pressure from the molars to the anterior teeth. This condition changes the physics of the masticating muscles from a second class lever to a third class lever.[11] The change in this application of force flattens the gonial angle of the jaw which results in the protrusion of the chin and backward pressure on to the tympanic plates. The question of focal infection can be omitted with this history, but the amount of irritation inflicted on branches of the fifth, seventh, ninth, tenth and eleventh cranial nerves is an unknown factor. Her profile showed thickened lips, drooping corner of the mouth, flabby facial lines, and the change in neck contour that is present in these cases.

Through raising her bite 4 mm. and putting biting pressure back in the molar regions her condition improved as follows:

Her physician had been treating her twice weekly for congestion of the nasopharynx, general debility and lack of interest in life. Her resistance was so low that automobile rides caused excessive fatigue.

New dentures were inserted in January, 1935. The physician's visits became unnecessary. By spring she was riding 100 miles comfortably, her mental outlook responded and she spent the following summer with a renewed outlook on life. Her appearance became greatly improved.

May the day soon come when the need for functional occlusion will be universally recognized, and when the extraction of teeth without replacement will be considered detrimental to the maintenance of oral health.

Osler, many years ago, recognized the importance of this subject when he said: "There is not one single thing in preventive medicine that equals mouth hygiene and the preservation of the teeth."

SUMMARY

Costen and others have shown that reflex disturbances from a pathologic temporal-mandibular joint are not uncommon.

As the normal function of this joint is dependent upon the proper occlusion of the teeth it becomes necessary to have a close cooperation between otolaryngologists and dentists to correct the reactions caused by disease in this area.

The diagnosis of an abnormal function existing in the joint is quite simple, but the re-establishment of occlusal equilibrium necessary to restore the joint to health is a serious dental problem.

The technic necessary to restore function permanently requires much skill, time and expense, and unless relief is assured in advance few patients are willing to undergo treatment.

Through the use of temporary adjustable splints, however, normal relations between the teeth and jaws can be quickly established and oral function restored. If the reflex disturbances improve, permanent restorations can then be constructed.

Advice should not be given too freely to the patient by anyone to the effect that the maintenance of normal function in this joint is a simple dental problem through the use of any type of treatment.

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XIII

MALIGNANT DISEASE OF THE SINUSES AND NASOPHARYNX, IN THE SMALL HOSPITAL*

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WATERVILLE, MAINE

This paper will attempt to discuss the problem of malignant disease of the paranasal sinuses and nasopharynx from the viewpoint of the clinician working in a small hospital, not having immediately available all the obvious advantages found in the large staff of a teaching medical center. In the otolaryngologic literature of the past few years there have been a number of valuable papers on this subject, written by such authorities as New, Beck, Barnes and others. From these one may obtain statistics and conclusions which would make any like attempt here seem preposterous.

The frequency with which the otolaryngologist encounters these cases depends somewhat upon his diagnostic acumen, and whether or not he keeps always in mind the possibility of malignancy. What should be the attitude of the otolaryngologist in the small hospital toward these cases? Should he be satisfied with making the diagnosis and then refer the case to some large medical center, thereby relieving his mind of much worry? Possibly so, if he is situated conveniently near. Otherwise it seems necessary for him to shoulder the burden himself, making the best use of available facilities and endeavoring to remedy existing omissions. New,[1] stressing the need for continued observation for a long time in these cases, stated that it would be better if they could be taken care of at or near their homes. This is further emphasized by the fact that these cases seem more likely to occur in persons in poor financial circumstances, where the expense of a long absence from home is sometimes prohibitive. This factor is well recognized by the American Society for the Control of Cancer and is one reason for its activities in endeavoring to establish tumor clinics at strategic points throughout the country. Bearing in mind the incidence of cephalic location of malignancy, would it not be advisable to include an otolaryngologist in the personnel of each such tumor clinic?

*Read before the Eastern Section of the American Laryngological, Rhinological and Otological Society, Boston, Mass., January 7, 1937.

The purpose of this paper is to point out certain errors encountered in the management of malignant disease of the sinuses and nasopharynx, and the most obvious handicaps to the successful handling of these cases in the small hospital; and finally to suggest some means to remedy them. A few selected case reports, illustrative of certain points, will be given.

Technic is not properly within the province of this paper and, except for a few broad generalizations, will not be discussed. It is assumed that all cases will be thoroughly studied preoperatively and all indicated laboratory and x-ray examinations done. In these cases avertin, sometimes reinforced by nitrous oxide and oxygen, has been the anesthesia of choice. The use of the cutting and coagulating currents, as described by Beck,[2] has been used in all except the earliest cases. Even the undifferentiated type of nasopharyngeal growths have been coagulated, followed by radiation. This has been done because our roentgenologist, Dr. J. P. Goodrich, feels that if accessible tissue is first destroyed, the radiation problem with that remaining is simplified. Usually the modified Moure incision, kept open for inspection as suggested by Barnes,[3] has been used in the sinus cases, although with orbital involvement this route has been employed.

The commonest error is that of failure to make a clinical diagnosis. This far outweighs in importance the often suggested failure of the patient to consult the physician early enough. Too often the story is that they have been treated for months with futile therapy, or been subjected to needless operations, without any conception of the possibility of malignancy, when a valuable clew was present in the history alone. Family history may be most suggestive. Likewise a story of persistent bleeding, bloody discharge, external swelling or tenderness, especially if unilateral, should keep the possibility of malignancy in mind.

REPORT OF CASES

CASE 1.—Mr. D., age 37. Right intranasal antrum operation four years before present admission. Several attacks of severe sinusitis in the interim. History of cancer on both sides of the family. Swelling of right side of face and increased nasal obstruction and discharge for several weeks. Examination showed firm indurated swelling underneath orbit and a patent operative opening into the antrum, which bled on being probed.

Operation, using the cutting and coagulating currents, revealed the outer antral wall missing, the antrum filled with freely bleeding neoplastic tissue extending into the ethmoids, and the roof and posterior bony wall over the pterygoid fossa missing.

Pathologic report, "Adenocarcinoma, grade III."

Fig. 1. Case 1.

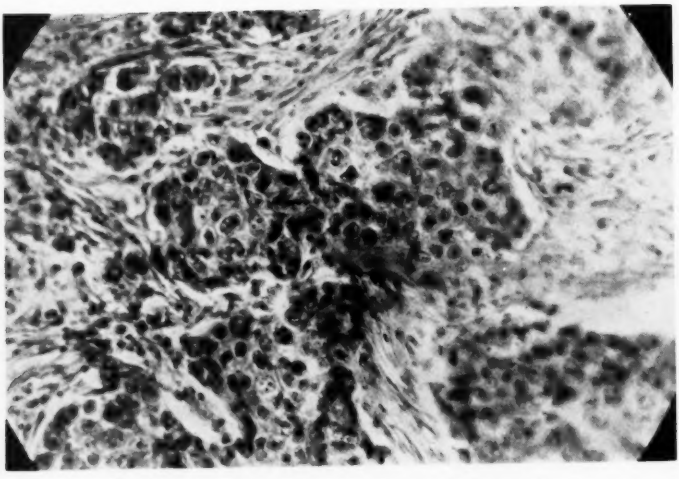

Fig. 2. Case 1. Adenocarcinoma, Grade III. 300 x.

One subsequent removal of tissue, using the same technic, was required. Three thousand mgm. hours of radium were given postoperatively. There have been no signs of recurrence in almost five years and, except for the operative deformity, he is perfectly well.

Failure to consider the nasopharynx carefully in cases of conduction deafness due to tubal obstruction, of trismus, of involvement of cranial nerves and of cervical adenopathy, has often caused delay in the recognition of nasopharyngeal malignancies. While many of these are quite radiosensitive, metastases to the cervical glands are apt to be very resistant to radiation, so that the prognosis, never too encouraging, is made much worse by delay in diagnosis. Too many cases are treated with never a careful examination of the nasopharynx. The nasopharyngoscope is indispensable in these cases.

CASE 2.—Mrs. B., age 55. Patient referred by one of the leading otologists in the country, now deceased, with a diagnosis of "acute secretory catarrh in the left ear" and a request that I catheterize her eustachian tube. The nasopharynx contained a mass of easily bleeding tissue which overlapped the tubal orifice. Biopsy was advised but the patient refused and left my care. Later she consulted an internist, because of enlarged glands in the neck, axillæ and groins. A diagnosis of Hodgkin's disease was made. Subsequently a gland was removed for microscopic study and a report of "lymphosarcoma" was made. Patient died shortly afterwards. Undoubtedly the primary lesion was in the nasopharynx.

CASE 3.—Mr. C., age 54. Gradually increasing deafness of the conduction type in the right ear for three months. There was a good sized preauricular gland just over the parotid. The nasopharynx contained a small amount of soft, easily bleeding tissue, filling the right fossa of Rosenmüller.

Biopsy reported as "epidermoid carcinoma."

As much as possible of the growth was destroyed by electrocoagulation. This was followed by intensive x-ray therapy, over a period of several months. While the patient seemed to improve for the first five months, this improvement was more apparent than real, for shortly he began to fail and died ten months after treatment was first instituted.

CASE 4.—Mr. S., age 17. Gradually increasing deafness in the left ear for eight months. Had been treated with inflations and had a septum operation performed elsewhere. There was a typical tubal occlusion with a conduction type deafness. There was trismus and tenderness in the left mandibular joint. Nasopharyngoscopy showed a mass of what was apparently adenoid tissue impinging upon the orifice of the left eustachian tube.

Pathologic examination of this was reported as "normal adenoid tissue." This diagnosis seemed open to doubt, and soon the increase of symptoms made this evident. He was reoperated upon and as much as possible of the tissue removed with the cutting current and the base deeply coagulated. This tissue was sent to another pathologist, who reported, "Lympho-epithelioma, highly radiosensitive."

Following this he was given intensive x-ray therapy. There has been no signs of recurrence in the nasopharynx in over two years and his hearing has returned

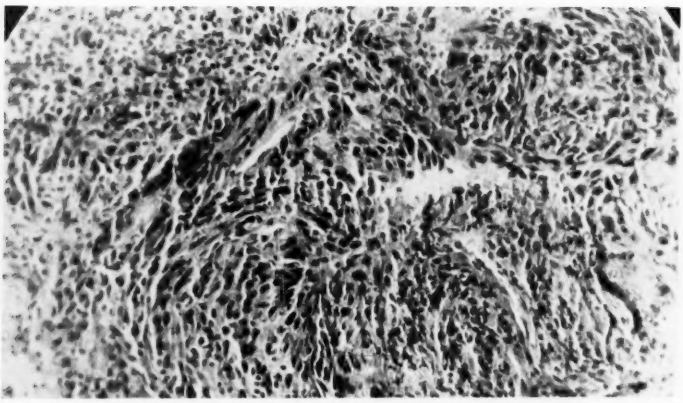

Fig. 3. Case 3. Epidermoid carcinoma. Not particularly well differentiated but definitely epidermoid rather than the so-called transitional type, or lympho-epithelioma. Would not expect it to be very radiosensitive. 330 x.

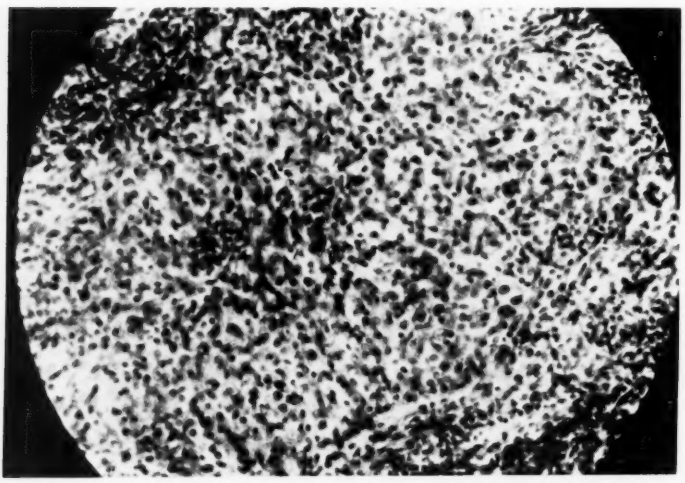

Fig. 4. Case 4. Lympho-epithelioma. Highly radiosensitive. 280 x.

to normal. He has gained sixteen pounds in weight and felt well except for the development of enlarged metastatic glands in the right side of the neck, posterior to the sternomastoid. While these have been quite resistant to x-ray therapy, they have now subsided and seem apparently under control. Of course, the prognosis in this case is rather poor. Earlier recognition of the nasopharyngeal neoplasm and eradication before metastasis occurred would have been advantageous.

Failure to carefully follow cases postoperatively and to thoroughly investigate any suspicious condition occurring perhaps years after operation for what was then a benign condition is another possible source of error.

CASE 5.—Mrs. M., age 43. Submucous resection of septum twelve years previously. For several months had had left-sided obstruction, scabbing and bleeding. Examination showed an area of easily bleeding, exuberant tissue on the left side of the septum about opposite the middle turbinate.

Biopsy reported as "carcinoma, hair matrix type."

This was completely removed with the cutting current, resulting in a large perforation of the septum but no recurrence in five years.

With the more universal acquaintance natural to a smaller community, there may be a disinclination to be as radical as is necessary, especially with women patients who object to facial disfigurement. This is a handicap that the operator in the large center, with an absolutely impersonal attitude, is not apt to encounter. It usually takes a few sad lessons to enable oneself to be as ruthlessly radical as the case demands.

CASE 6.—Mrs. W., age 47. Pain and tenderness in the right side of the face, nasal obstruction and bloody discharge for several months. The right side of the nose was filled with a freely bleeding polypoid mass. The inferior turbinate was atrophic. Operation performed through canine fossa in order to avoid deformity, although it was realized we were dealing with a malignancy. The outer antral wall was eroded, as was the nasal wall. There was porky tissue infiltrating the cheek. The antrum was filled with bleeding neoplastic tissue.

Pathologic report, "Adenocarcinoma, grade 11." Three thousand mgm. hours of radium was given postoperatively. Patient died in a few months. There should have been no excuse for anything short of a radical external exposure in this case. This was one of the earlier cases and was not done with the cutting and coagulating currents, which was an additional handicap.

Another possible source of error in small hospitals is failure to insist upon a pathologic examination of all tissue removed at operation. It is a good rule in any hospital that all such tissue go to the pathologist, at least for macroscopic examination, and that all tissue other than routine material, obviously without suspicion, be studied microscopically. Omission of roentgenologic examination is another frequent cause for error.

Fig. 5. Case 7.

CASE 7.—Mr. P., age 50. Right-sided nasal obstruction, pain and tenderness for six months. Polyp removed a few weeks before by a physician in another town. This was not examined. Following removal he had a marked increase in symptoms and was referred to our hospital.

X-ray examination showed "antrum opaque, roof practically destroyed, with extension of destruction through anterior ethmoids." Biopsy reported as "epidermoid carcinoma, grade III."

Operation disclosed the outer wall and roof of the antrum completely eroded, porky tissue infiltrating the cheek and filling the antrum and ethmoid region, and the posterior wall of the antrum missing, exposing the fascia of the pterygoid fossa. Three thousand seven hundred and fifty mgm. hours of radium given postoperatively, followed by x-ray therapy. The patient has been well without signs of recurrence for almost two years. If the first physician had had an x-ray examination before operating, I doubt if he would have done so futile a procedure. Careful examination of the "polyp" might likewise have given valuable information.

One of the greatest handicaps is that all pathologists are not well versed in respiratory tract pathology. This is more apt to be the case in the small hospital, where only a fraction of the pathologist's time is devoted to microscopy. Often he carries on his laboratory work as a part-time procedure. And even the best of pathologists are subject to errors, just as are clinicians. Nash,[4] in his paper two years ago, showed how leading pathologists might disagree on microscopic diagnosis. It seems the wise thing to check on the pathologic diagnosis when the clinical observations indicate some discrepancy, just as we have no hesitancy in asking for consultations on our clinical prob-

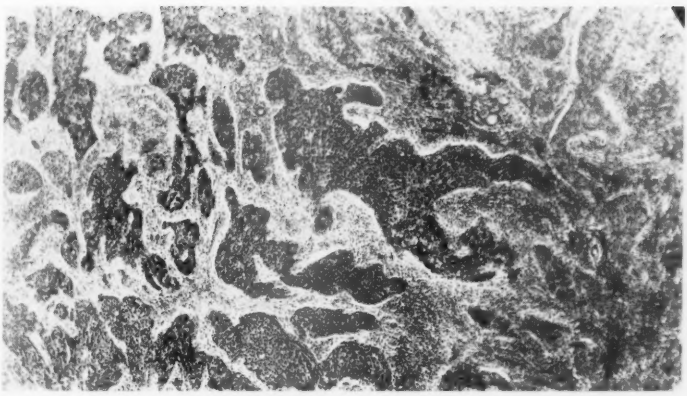

Fig. 6. Case 7. Epidermoid carcinoma, grade III. Tumor fairly well differentiated but is growing so rapidly and shows so many tumor giant cells that it must be regarded as highly malignant. Small foci of mucinous degeneration are noted, the significance of which is not understood. 75 x.

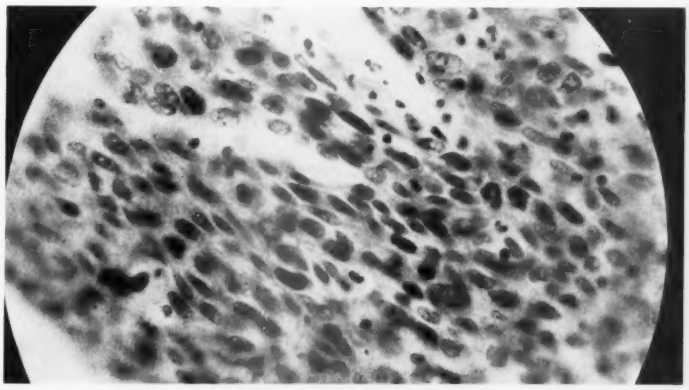

Fig. 7. Case 7. Epidermoid carcinoma, grade III. Tumor fairly well differentiated but is growing so rapidly and shows so many tumor giant cells that it must be regarded as highly malignant. Small foci of mucinous degeneration are noted, the significance of which is not understood. 630 x.

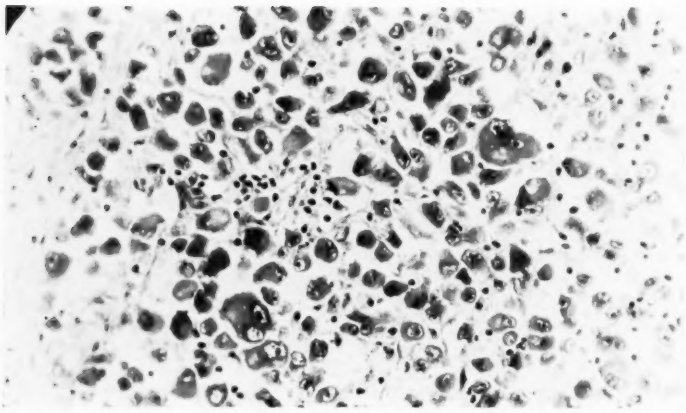

Fig. 8. Case 8. Malignant tumor, unclassified. A poorly differentiated, rapidly growing and highly malignant tumor. A definite classification cannot be made, but it is consistent with an undifferentiated carcinoma, although fibrosarcoma cannot be ruled out. 350 x.

lems. This is no reflection on the pathologists. Easily they pick up more of our errors than we ever will of theirs.

CASE 8.—Mr. K., age 49. Right-sided nasal obstruction for some time. Ten months before, a polyp had been removed by a physician in another town, who evidently suspected malignancy, as he sent the patient to a well known metropolitan hospital, where a diagnosis of "melanotic sarcoma" was made. An intranasal operation was performed, using the coagulating current, and the patient was discharged home. Since then he had not been under medical observation. Examination showed the right side filled with a mass of necrotic, friable, bleeding tissue. Externally there was a small tumor just below the inner canthus.

Biopsy was reported as "malignant tumor, unclassified. The external tumor is a typical basal cell carcinoma."

Operation, using the cutting and coagulating currents, showed that the outer antral wall had not been invaded, although the nasal wall was practically gone. He was given 3,600 mgm. hours of radium. Following this there were no signs of recurrence in the nose, but three months later he developed metastatic glands in both sides of the neck. X-ray therapy seemed to have no effect. Radical removal of these glands was now done, attacking one side at a time.

Pathologic report from the right side showed "an extreme degree of rapidity of growth and lack of differentiation. It strongly resembles an embryoma of the testicle. It might well prove radiosensitive." The report from the left side was as follows: "Comparison with the previous specimens . . . . shows that all are of closely

similar structure, so that there can be no doubt of a common origin. The cells are loosely adherent, practically without stroma. They are polyhedral, have vesicular nuclei with multiple nucleoli, a dense slightly acidophilic cytoplasm. Tumor giant cells with multiple nuclei are numerous and mitotic figures frequent."

Following this he had intensive x-ray therapy. There was no evidence of recurrence on the left side, but after four months marked metastasis occurred on the right. When seen at this time there was an ulcerating mass the size of an orange in the posterior triangle, as well as a large tumor in the axilla. In purely a palliative attempt, the mass in the neck was removed and further x-ray treatment given. The patient died one month later. The prognosis was probably hopeless from the start, yet earlier and more intensive attention would have been preferable.

Under conditions where the best microscopy may not be immediately available, it is quite possible to arrange to have this done at one of the large hospitals where there is a full-staffed pathologic department. This is the procedure we have adopted on all microscopy except where frozen sections are demanded. We feel it assures us of authoritative opinions. With air mail service as it is, this can be done with little more delay than in our own laboratory.

Another handicap, which may be fairly well overcome, is the difficulty of getting expert advice as to postoperative radiation. Naturally the experience of our men working with radium is somewhat limited, although it is possible to get very satisfactory roentgen therapy. I have always found the leading authorities on radiation most helpful and willing to advise as to dosage, basing their advice upon the history and microscopic slide. While, of course, this method is somewhat of an imposition upon them, the whole management of these cases is usually a labor of love anyway, and everyone seems willing to do his part.

As stated before, any conclusions in such a paper would seem out of place and so will not be attempted.

SUMMARY

Malignant disease of the sinuses and nasopharynx can be handled fairly effectively in the small hospital. The commonest errors encountered are mainly due to failure to make correct diagnoses, overlooking the nasopharynx as a possible site of the primary lesion, neglecting to have proper roentgenologic and pathologic studies routinely made and to a misguided zeal to preserve the cosmetic result at the expense of being sufficiently radical. These are not attributable to the hospital but are personal factors, easily remedied. The greatest handicaps in the small hospital are the inferiority of the microscopy and the lack of expert advice on postoperative radiation. With the further development of tumor clinics throughout the country these

handicaps will become less of a factor and more progress will be made in the control of cancer.

The microscopy and pathologic diagnoses on the later cases, C, S, P, K, was done by Dr. Tracy B. Mallory, Massachusetts General Hospital, Boston.

The writer wishes to express his appreciation to Dr. Douglas Quick, New York, for his valuable advice regarding postoperative radiation in several of these cases.

PROFESSIONAL BLDG.

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XIV

WHY I HAVE NOT USED IONTOPHORESIS*

GORDON F. HARKNESS, M.D.

DAVENPORT, IOWA

I have been asked why I do not use iontophoresis for nasal allergy and vasomotor rhinitis. In formulating a reply I have attempted to reach my conclusions, which seem valid to me personally, without any bias that may hamper me in whatever future procedure I may wish to adopt.

First, to my regret, I realize that I am no longer a youngster in the practice of otolaryngology, but my years in medicine have seen temporary furores fill our literature and then fade away to an early oblivion. I have gone through a period of radical intranasal and extranasal surgery that was not based on sound surgical principles and which disregarded the essentials of nasal physiology. I have made an earnest effort not to become a therapeutic nihilist. I have concluded that, as far as I am concerned, with my patients, who are private patients, I am willing not to be among the first to adopt many procedures which blatantly confront me in our literature. With institutional clinical opportunities I would not wish to adhere so closely to such a policy but would want to experiment with new ideas.

Iontophoresis is not new. It is having an evangelistic revival. My experience with it in chronic suppurative otitis media, not comparable with this discussion, was most disappointing. Yet our literature gave many glowing accounts of its accomplishments. This may have prejudiced me.

Iontophoresis in nasal allergy and vasomotor rhinitis can lay no claim to overcoming a basic cause, but is an attempt to merely relieve a distressing symptom. This is not the desideratum of modern therapeusis. Its revival in this country was started largely by the reports of Warwick. This was so intimately associated with the promulgation of expensive commercial apparatus that I, for one at least, looked askance. A secret formula likewise added to the prejudice.

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*Adapted from a discussion before the Midwestern Section of the American Laryngological, Rhinological and Otological Society, Chicago, Jan., 1937.

I still remember my rather elaborate investment in chlorine producing machines which have been discarded, and the common cold still remains with us. I think of the money that hospitals and physicians have paid for complicated and expensive oxygen tents, when one knows that a nasal tube and a regulating valve are equally efficient.

Iontophoresis is based on simple laws of electrophysics. Amperage, voltage and ohms are not mysterious factors to be determined only by the most elaborate machines. Smooth currents likewise may be obtained in various uncomplicated ways. If resistance is too high, why stress the reapplication of moist pledgets in a normally moist cavity, rather than thinking first of epidermal resistance? If such care as to resistance and amperage is so essential, why disregard the varying sizes of nasal chambers?

Whether or not it accomplishes its purpose, my next inquiry is what harm can it do? McMahon has shown in his experiments with dogs a fibrosis of subepithelial tissue. Alden has shown a regeneration of epithelium with cilia. Hollender has shown a regeneration without cilia. Animal experimentation is not conclusive proof that human tissues will react similarly. More important to me is not the presence of a fibrosis or the absence of cilia, but what is the clinical effect of iontophoresis on normal nasal physiology? What about the humidity of air inhaled? Are such treatments followed by discomforts on the part of the patient? I have failed to find disinterested studies from this viewpoint. Where do cadmium and tin enter the picture? And, if so important, why does an independent analysis, as made for Hurd, show the electrodes failing to conform to the published claims? What of anosmia? Anosmia is a real affliction. What are the probabilities of its occurring, and if it does occur will it be temporary or permanent?

The personal equation enters into what reactions are necessary. Are the glary mucoid plugs, blocking the nose with marked discomfort for several days, necessary? Most advocates say yes, yet Hollender says no.

The personal equation also affects what constitutes relief and materially influences statistical reports.

Turning to the literature of the past three years, my bibliography includes thirty-six articles.

Not as an umpire, for he must know the rules of the game (and one side or the other always wants to kill him), but as a side line

spectator one is quite forcibly struck with the variations in results obtained and the source of these reports.

Rhinologists, immunologists, allergists and electrotherapeutists offer striking examples of the fact that something is wrong with medical statistics. The rhinologist has seen the allergist displacing him in relieving nasal symptoms, and his reports for seasonal allergy (pollenization allergy), speaking generally, are most favorable for relieving this condition. The allergist has tests and injections for these patients, but none for vasomotor rhinitis. His reports decry the use of iontophoresis for seasonal allergy but concede its position for hyperesthetic rhinitis. I do not mean to intimate that there is any deliberate distortion, but it does make one feel that some subconscious wish may be father to the thought.

Let me cite a few reports:

Harold Hays (Med. Rec., 141:23-25, Jan. 2, 1935) reports excellent results in four out of six cases of hay fever and five out of seven cases of hyperesthetic rhinitis.

S. W. Garfin and S. W. Pearl (New Eng. J. of Med., 214:244-47, Feb. 6, 1936), thirty-three out of thirty-five cases of hay fever and vasomotor rhinitis failed of complete relief, but eighteen out of twenty cases of asthma were relieved.

Sidney H. Gidoll (Cal. and West. Med., 40:187-188, March, 1934), twenty-seven out of forty cases of nonallergic sinusitis relieved.

G. W. Walker (Cal. and West. Med., 42:263-264, April, 1935), twenty-four out of twenty-eight cases of hay fever relieved.

B. L. Bryant (Jour. of Med. Acad. Med., Cincinnati, 15:598-601, Feb., 1935), seven out of ten cases of hay fever and hyperesthetic rhinitis received complete relief.

A. B. Hollender and M. Gorin (Ill. M. J., 68:493-498). Superior to all forms of treatment for vasomotor rhinitis, not a cure, but has an alterative effect. Hollender (Arch. Phys. Ther., 16:359-362, June, 1935) says iontophoresis is only occasionally effective in seasonal hay fever.

W. E. Warwick (Laryngoscope, 44:173-181, March, 1934) claims complete relief in thirty-one out of forty patients.

H. L. Alexander and J. W. Alexander (J. Allerg., 6:240-243, March, 1935) state that from the clinical standpoint iontophoresis is

an inferior type of treatment for hay fever, asthma and other allergic conditions but has a definite place in vasomotor rhinitis without established etiology. They showed that skin reactions, passive transfers, eosinophiles in the blood and secretions are not particularly affected and that relief was in reverse proportion to the number of reagins present.

L. B. Bernheimer (J. A. M. A., Vol. 106, No. 23:1980-1981, June 6, 1936) claimed the histologic effects were the same as from escharotics, with only 5 per cent of hyperesthetic rhinitis cases being favorably influenced and 100 per cent failure in altering the clinical course of hay fever.

Maximilian A. Ramirez, from the Department of Immunology of the French Hospital, New York City (J. A. M. A., Vol. 106, 4, p. 281, Jan. 25, 1936), reports the vast majority of twenty-five cases of nonspecific perennial vasomotor rhinitis were improved, but 100 per cent failure with fifty cases of seasonal pollinosis.

The group tabulation made by Alden (Laryngoscope, Vol. xlv, No. 8, 620-630, Aug., 1935) came from otolaryngologists. Roughly, success was obtained in the majority of hay fever patients in 75 per cent of perennial allergic rhinitis cases, and in about 25 per cent of allergic asthmatic patients.

Lee M. Hurd (Arch. Otolaryn., Vol. 22, No. 4:416-424, October, 1935) reports nearly all of his cases of hay fever and the majority of his hyperesthetic rhinitis patients were relieved. He published the table compiled by the Burdick corporation; relief in hay fever, about 75 per cent; hyperesthetic rhinitis, 60 per cent, and in allergic asthma, 45 per cent.

H. G. Tobey (ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. 44, No. 1, 95-97, March, 1935) reports relief in 85.6 per cent in hay fever, yet feels desensitization is the method of choice. His immediate results in vasomotor rhinitis were excellent.

To me, allergy at the present time is a good deal like a ship without a rudder. Some day, when someone discovers that primary basic fundamental something which must be common to all allergies, we will have the rudder that will take us out of this chaotic sea of individual sensitiveness to this and that. The cause of the discomfort from sensitized nasal mucous membranes has not been explained.

Iontophoresis is a local, symptomalogic treatment (general desensitization not having been proven), a form of cauterization perhaps, and may find a permanent place in our armamentarium. Time will

establish that. But I do offer a protest against current medical literature. Here we have a simple procedure, a definite symptomatology, and the easiest possible facilities for interpreting results. Individual technic should not be a material factor. Yet our literature gives the relief obtained varying from 0 to 100 per cent. And this we call scientific medicine.

507 PUTNAM BLDG.

XV

ULTRA SHORT WAVE CURRENTS IN THE TREATMENT OF EAR, NOSE AND THROAT CONDITIONS*

ALEXANDER F. LASZLO, M.D.

NEW YORK

The use of diathermy did not prove very successful in nose and throat practice except for surgical diathermy (coagulation); the short wave and ultra short wave machines were recently brought out as an improvement.

Three to four years have past since the first reports on the use of the short wave machines appeared.[1] The reports dealing with nose and throat conditions are meager,[2] but the few that are found in the English literature are quite enthusiastic.[3 4] That the method is not without danger and the machines cannot be used without undue care appears from the articles recently reported by different American authors, through the investigation of the Council of Physio-Therapy, calling attention to the mishaps and accidents that have occurred while using these machines.[5 6 7]

To enable us to form an unbiased opinion, we selected 100 ear, nose and throat cases and undertook their treatment with ultra short wave therapy. I do not intend to go into the technical part of these machines that are so ably described in the latest textbooks.[6]

The majority of these cases were sinus infections, thirty of which were chronic and had resisted all therapeutic and operative measures at our command. Thirty were acute and subacute sinus conditions with and without pus formation. The others were laryngitis, lymphoadenitis, furuncles of the ear and nose, peritonsillar abscesses, acute and chronic middle ear conditions and acute and chronic mastoids.

In the majority of these cases I used spark-gap machines with changeable wave lengths (mostly six meter), with 150 watt output. On the other cases a machine of fixed gap (9½ meter), with 150 watt output, and a tube machine (6 meter), with 400-watt output was used.

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The duration of each treatment was adjusted to the tolerance of the patient. At the beginning children were treated for five minutes, and adults ranged up to 20 to 25 minutes, with an average of 10 to 15 minutes. The majority of the patients were treated lying on a wooden table, with the electrodes on felt and turkish towels applied directly to the head. Other patients sat on a wooden chair, with the electrodes resting on a special stand, with glass and air spaces between the electrodes and the part of the body under treatment.

GENERAL OBSERVATIONS

Almost all the patients were satisfied with these treatments, and even small children, after a treatment or two, gave no difficulty. Patients, who received both types of treatment, preferred the treatment with the electrodes attached to the treated part. The application of the electrodes to the head was difficult, due to the prominence of the nose. I applied them in the anterior-posterior position, and later to the sides of the face or the head. In the beginning I used specially devised sinus electrodes, but their value could not be demonstrated.

The patients were under continued supervision during the treatments. Probably this is the reason no untoward effects were observed, although many thousand treatments were administered.

In about twenty cases I measured the temperature before and after the treatments, and no change was noted. In the same number of cases the temperature was taken while under treatment. Specially constructed thermometers were applied to both nostrils. The nose was plugged with cotton to prevent the air circulation through the nose. No considerable rise in temperature was noticed under either method used. Measuring with the latter method, an increase of .3 F. was noted. In cases where breathing through the nose was permitted during the treatment, absolutely no rise in temperature was observed. In none of the cases were thermometers inserted into the sinuses. Taking the temperature by mouth gave exactly the same result.

There was local sweating with almost every patient, but this was absorbed by towels applied to the skin. Every patient felt very comfortable, and many of them did not want the treatments to be discontinued. In the cases treated with anteroposterior positions there was an occasional redness of the tip of the nose, but this disappeared a few minutes after treatment. Later on I used an extra protection of absorbent cotton on the nose.

Every patient felt the heat for a little while after each treatment, but none of them experienced it for a long period. When pa-

tients complained of a headache at the beginning of the treatment, I would immediately discontinue it. With a slow increase in the length of time in sensitive patients, this complaint seldom reappeared. A few, especially the older patients, complained of a passing dizziness, which in two cases was quite severe; but a few minutes' rest returned them to normal.

A lowering of the blood pressure was observed in almost every case of treatment of the head. This symptom was slight and passing in patients with normal blood pressure, but sometimes quite severe, reaching a drop of almost 40 mm. in cases of blood pressure around 200. These experiments were repeated and checked by others, but in no case did the effect last longer than twenty minutes.

Blood counts before and after the treatment showed nothing of significance. There was a slight leucopenia immediately after the treatment and a slight leucocytosis a few hours later. Although the majority of the cases followed this rule I could not find it in every case.

The number of treatments given ranged from six to thirty-five or forty, depending on the case.

Among the sinus conditions treated with short wave therapy were infections of the maxillary antrum. The majority of these antra were shown by lavage to contain free pus, and all had clinical symptoms and x-ray changes. The acute cases received the most benefit, as the pain disappeared and the clinical symptoms were ameliorated. In some cases this benefit lasted for several weeks. None of these antra cleared up entirely. Although the clinical symptoms were relieved they still contianed pus after as many as twenty-five treatments, whereupon we had to return to washing the antra and occasionally to surgical intervention. In those cases where infection was manifest only by postnasal drip, unproductive cough, obstruction and tightness of the nose, very little relief could be observed and there was only temporary subjective improvement.

Acute and sub-acute purulent ethmoiditis with pain reacted well. I observed a few fulminating cases of acute ethmoiditis, some of which were in children, with and without external edema. The pain disappeared entirely after a few treatments and the edema about the orbit subsided, but the discharge still remained and some of the adults had to undergo surgical treatment. Cases of chronic ethmoiditis with hyperplastic changes received no benefit whatsoever. Sphenoid infections with postnasal drip and headaches received benefit only in as much as the headache was concerned.

The cases of acute frontal sinusitis treated by ultra short wave therapy also received cocain-adrenalin tampon applications in the middle meatus. Beside this the patient was advised to rest and apply heat to the sinus while at home. The difficulty in evaluating the treatment lies in the fact that the majority of these cases react well without the short wave therapy under similar circumstances. Acute colds with coryza and rhinopharyngitis were seemingly shortened by ultra short wave therapy. Some of the vasomotor rhinitis cases reacted well, at least temporarily, while some did not respond. A few cases of acute laryngitis were treated with good results, but aside from the short wave treatments, general care and rest of the voice was emphasized to these patients. There was no improvement in three cases of chronic laryngitis.

The best results were obtained in superficial infections of the ear, nose and upper lip, and throat (peritonsillar abscesses). In furunculosis of the ear the pain was diminished and all the cases cleared up after one or two treatments. Infections treated in the beginning stage regressed, while in the more advanced stage the pointing hastened and the rupture was spontaneous. In furuncles of the nose and upper lip, where surgical intervention should be avoided if possible, ultra short wave therapy is effective. Peritonsillar abscesses in the beginning stage usually disappeared after one treatment. In the more advanced cases the pain was diminished and pointing was hastened. The disappearance of the pain allowed the patient better to take nourishment and the characteristic rigor was lessened.

Lymphadenitis with persistent high temperature, especially in children, received great benefit after a few treatments. In none of the six that we treated was there any necrosis.

Nonsuppurative inflammations of the ear and eustachian tube were shortened by ultra short wave therapy. In cases of acute purulent otitis media paracenthesis was preferred. Two cases of mastoiditis were treated. One of them got much worse after the second treatment and was operated on, while the other cleared up without operation after the fifth treatment.

Many cases of chronic otitis media, suppurative and nonsuppurative, received treatments. There was no indication of improvement in any of these cases, nor in any of the various types of deafness.

SUMMARY

The number of cases reported and the time elapsed are not sufficient to draw definite conclusions. We are still ignorant of the pre-

cise action of these machines. Our experience with heat in other forms is encouraging and here we have a new method of applying it. The results obtained are based more on clinical impressions than pathologic investigations. The surgical principle of adequate drainage should not be forgotten and short wave therapy should be utilized only in selected cases.

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XVI

ORAL AND PHARYNGEAL MANIFESTATIONS OF DERMATOLOGIC CONDITIONS*

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It is impossible in a single paper to consider all of the dermatologic diseases which involve the oral and pharyngeal regions. The otolaryngologist is entirely familiar with many of the more common conditions, such as syphilis, tuberculosis, leukoplakia, neoplasms, the exanthemata, and with conditions such as Vincent's symbiosis and canker sores, which are limited to the mucous membranes. Other conditions, such as the rare occurrence of keratotic lesions in the mouth in cases of keratoderma blennorrhagicum (gonorrhea[2]) can only be mentioned by name in a paper of this type.

Cutaneous blastomycosis (Gilchrist), either in its localized or systemic forms,[30] is seen particularly in this part of the country. The localized type occurs anywhere on the glabrous skin, but it most frequently occurs on the face as sharply defined, elevated, verrucous plaques which tend to clear in the center and spread peripherally. With a hand lens at the periphery of the lesion, one can see minute abscesses, and by expressing the contents of these abscesses, Blastomyces can be demonstrated in preparations of potassium hydroxide and also in cultures. Direct extension may occur to the lips and mucous membranes of the mouth (Fig. 1). Several cases of blastomycosis of the larynx, pharynx, and trachea have been reported[15,25]; in some of these cases there was no evidence of any lesions on the skin. These lesions, when they are situated in the oral cavity, pharynx, or larynx, do not differ clinically from those seen on the skin, although central involution may be lacking. Laryngeal obstruction may become pronounced enough to necessitate a tracheotomy. A case in which primary blastomycosis was limited to the esophagus has also been reported.[37] Repeated dilatations of the esophagus were necessary; following these, the patient responded satisfactorily to the ad-

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Fig. 1. Blastomycosis of the lip with involvement of the mucous membrane of the cheek.

ministration of potassium iodide and local applications of gentian violet. He finally died elsewhere, probably of the pulmonary tuberculosis, which had been previously demonstrated, rather than from the blastomycosis. Blastomycosis of the oral cavities, like the cutaneous forms of the disease, may at times be difficult to distinguish clinically from the verrucous forms of tuberculosis. Likewise, syphilis and neoplasms may resemble blastomycosis so that the taking of a specimen for biopsy may be necessary to make a diagnosis.

The Blastomyces is a double-contoured refractile organism, 7 to 20 microns in diameter, which in the tissue is seen to be dividing by budding rather than by endosporulation, as does the Coccidioides immitis. The histopathologic picture is also typical and reveals microabscesses which are situated within the epidermis and contain polymorphonuclear leucocytes. It also reveals single and budding Blastomycetes within giant cells, or occurring free in the tissue (Fig. 2). At times the large number of giant cells seen in a section suggests the diagnosis of tuberculosis, but there is no formation of true tubercles. The marked proliferation and thickening of the epidermis is not to be confused with true epithelioma. Persistent and intensive treatment with iodides in conjunction with radiotherapy or excision by the cutting cautery has resulted in apparent cure in some cases in which there were lesions of the oral cavity, as well as in many cases of local-

ized cutaneous blastomycosis. However, there is always danger of systemic dissemination of the disease from the local lesions in the skin or oral cavity, or the disease may spread from a focus in the lung or another internal organ to produce multiple abscesses and ulcers in the skin and mucous membranes.[22] Systemic blastomycosis almost always results fatally.

Coccidioidal granuloma, which is endemic in the San Joaquin valley in California but rarely originates elsewhere, may also result in ulcers or granulomatous lesions in the oral cavity.[42] When examined in histologic specimens or in smears treated with a solution of potassium hydroxide, the Coccidioides immitis appears as a double refractile organism which is much larger than the Blastomyces. It divides by endosporulation and not by budding. The clinical picture of coccidioidal granuloma may at times closely simulate that of blastomycosis, especially the systemic forms.

Both conditions may simulate torulosis (Cryptococcus hominis), which usually attacks the brain and meninges, but Jones reported a case of torulosis in which destructive granulomatous lesions were limited to the nasopharynx and apparently were arrested by treatment. Urbach and Zach described a case of generalized torulosis which included cutaneous lesions. In this case the disease began as a chronic proliferating gingivitis, with papillomatous and ulcerative lesions. This disease is caused by a double-contoured, refractile, budding organism similar to, but smaller than, the Blastomyces. With special stains, one will find a definite zooglea-like membrane around the organism. Wile emphasized that pathologically there is an absence of abscess formation, such as is usually seen in blastomycosis, and that in cases of torulosis a great many more organisms usually are present in the tissue. These organisms exist free or are contained in giant cells. The pathologic picture more closely simulates that of tuberculosis in its architecture. Definite distinction between blastomycosis, coccidioidal granuloma, and torulosis is usually dependent on demonstration of the causative organism by various laboratory studies.[22] Benham considered related but rare diseases in this group, such as the histoplasmosis of Darling, lymphangitis epizootica, and various forms of chrome blastomycosis. The various forms of so-called South American blastomycosis may also be associated with involvement of the oral cavity.

Sporotrichosis[31 33] may at times involve the mucous membrane, usually in the form of ulcers which do not as a rule extend as deeply as do those of tuberculosis and syphilis. Diagnosis is dependent on culture of one of the several genera Sporotrichum.

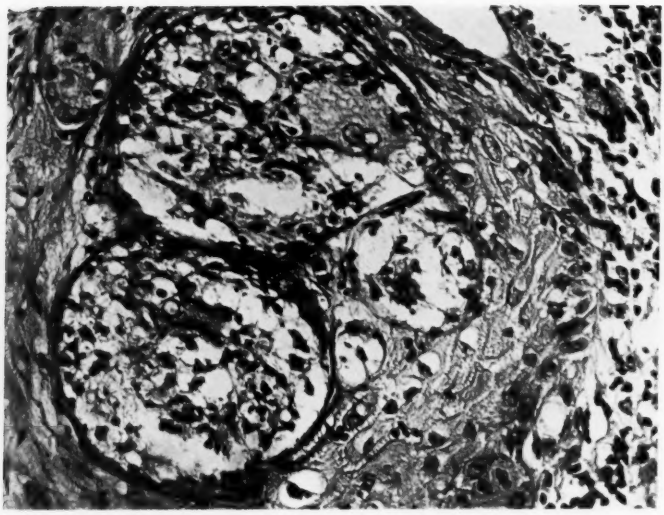

Fig. 2. Blastomycosis, showing intra-epidermal micro-abscesses containing polymorphonuclear leucocytes and giant cells. One may note double refractile budding Blastomyces in giant cell near top, and solitary Blastomyces in smaller giant cell in the lower abscess.

Actinomycosis of the tongue and oral cavity is a familiar subject and needs only be mentioned.[27] Demonstration of sulphur bodies and actinomycetes permits of a definite diagnosis.

McGovern has recently reported primary tularemic ulcers in the pharynx. These were characterized by large confluent ulcerations involving the right side of the posterior wall of the pharynx and by many discrete shallow ulcerations which were about 3 cm. in diameter and were covered with a gray exudate. The cervical lymph nodes were enlarged and tender; and fever was present. The patient did not respond to treatment for Vincent's angina. The infection resulted from picking ticks from the ears of a pet dog and crushing them between the fingers; the infection was carried to the mouth by the fingers.

Xanthoma is not uncommonly seen involving the mucous membranes of the mouth in association with cutaneous lesions of xanthoma tuberosum multiplex, xanthoma diabeticorum, and even xanthelasma. Lesions involving the pharynx, larynx, and trachea are less common,[11]

but it is possible that many have been overlooked in the past, especially lesions like those found in the case recently reported by New,[26] in which no cutaneous lesions were present. The clinical picture of xanthoma is easily recognized; the tumors appear as yellowish nodules, vary considerably in size, and often are grouped together to form yellowish plaques. In some instances they result in obstruction of the trachea, and even may necessitate tracheotomy. The histopathologic picture is diagnostic and is the same for all types of xanthoma (Fig. 3a). Stains for fat reveal xanthoma or foam cells laden with various lipoids, and large numbers of so-called Touton giant cells frequently are seen. These are characterized by central circular arrangement of their nuclei with a peripheral zone of cytoplasm which contains lipoid material. Xanthoma must not be regarded simply as a dermatologic condition; it also represents general systemic xanthomatosis which varies greatly in its manifestations, and includes such conditions as Gaucher's disease, Schüller-Christian's disease, and Niemann-Pick's disease. There is often a marked increase in all the blood lipoids and also a relative and absolute increase in the amount of cholesterol in the contents of the tumor as well as in the blood plasma.

Another condition in which foam cells are present is rhinoscleroma.[9] The otolaryngologist is familiar with the clinical picture of this disease; however, I want to emphasize a fact not generally acknowledged in the literature, namely, that the histopathologic picture of rhinoscleroma is diagnostic because of the following combination of findings: (1) the presence of Mikulicz's or large foam cells containing Frisch's encapsulated bacillus; (2) the presence of a great many plasma cells, even suggestive of a plasmoma, and (3) the presence of so-called Russell bodies (Fig. 3b). No other condition presents this combination of pathologic changes.

Specific or nonspecific lesions of the mucous membranes are not an uncommon finding in any of the lymphoblastomas.[23] This term includes mycosis fungoides, Hodgkin's disease, leukemias of various types, and lymphosarcoma. The lesions of the mucous membranes are most frequently seen in the acute forms of leukemia. They may be ulcerative, bullous, hemorrhagic or even necrotic in character, and may have to be distinguished from pemphigus, thrush, drug eruptions, and various granulomatous processes. Lynch has described hemorrhagic areas on the uvula and buccal mucosa, and others have described ulcers in monocytic leukemia of either the Naegeli or Schilling types. Lymphedema (elephantiasis) of parts of the face and the genitalia or of one or more extremities may result from involvement of adjacent lymph nodes by a lymphoblastomatous process.

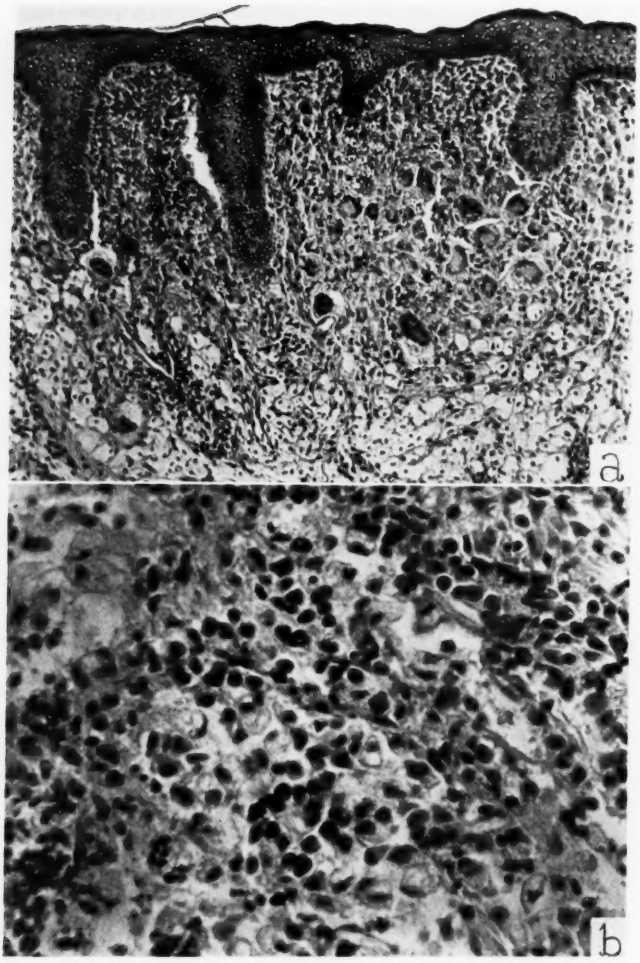

Fig. 3

3a. Typical xanthoma showing foam or xanthoma cells in lower portion, and numerous Touton type of giant cells (x 75).

3b. Typical rhinoscleroma showing Mikulicz's cells (foam cells), plasma cells, small black-staining Russell bodies and a few leucocytes in left lower corner (x 415).

Not infrequently it may result from a lymphoblastomatous infiltration of the lymphatic vessels which causes marked uniform swelling of the entire face. Striking but temporary response may be seen following radiotherapy. Diagnosis is dependent on histologic and hemocytologic studies.

New and Kirch have recently described a group of cases of permanent enlargement of the lips and face. The enlargement was secondary to recurrent swellings and was associated in many instances with facial paralysis. The condition may occur at any age, and the swellings come on rather suddenly without any signs of local inflammation. After the second or third attack the swellings remain and enlargement of the region increases. The inner aspect of the lips and tongue, as well as the nose and soft palate, may be involved in the process. The condition differs from angioneurotic edema in that the trouble is confined to the face, there are no remissions and no history of preceding lymphangitis or erysipelas is obtainable. In angioneurotic edema there is no permanent enlargement or facial paralysis. In the cases reported by New and Kirch, examination of the skin revealed a soft, nonpitting, smooth, diffuse enlargement, and histologic examination simply disclosed edematous tissue which contained some lymphocytic infiltrate. Satisfactory treatment has consisted of injection of boiling water into the involved parts and irradiation externally over the face.

Fungi may be the cause of several benign disorders in the mouth. So-called hairy tongue or black tongue is an example of this.[35] The tongue may be any color from light brown to black or blue, and the entire dorsum of the tongue is usually covered with an adherent furry material which resembles in appearance the mohair used for upholstery. The disease probably is caused by a genus of Actinomyces and usually causes no subjective symptoms. I know of no specific treatment for it.

Finnerud has demonstrated that perlèche is the result of infection by Cryptococcus or Monilia. The disease occurs especially among children, but it also may be seen among adults (Fig. 4).[13,34] It is characterized in the beginning by an ill-defined, smooth, grayish-white thickening on the mucous side of the commissures of the mouth. Later, there appears a bluish-white or pearl-colored thickening of both lips; there may be some wrinkling and occasional maceration, but there is never any ulceration. The infection is much less frequent among adults than it is among children. A deepened groove at the angle of the mouth and increased oral acidity, associated with oral fermentation, may be factors in producing a favorable terrain.

Fig. 4. Perlèche showing involvement of the corners of the mouth.

Treatment consists of local applications of 8 per cent solution of silver nitrate, twice a week, and the use of solutions containing gentian violet and other antiseptics.

Generalized systemic thrush of children, better referred to as moniliasis,[3] may present extensive cutaneous lesions, as well as marked involvement of the mouth. The whitish plaques on the mucous membranes appear as false membranes and usually are readily removed. In some cases in which stomatitis affects adults, the lesions simulate aphthous stomatitis and also are result of infection by Monilia. It is not uncommon to find at the same time ulcers and scaling plaques on the labia and vagina, which also are caused by Monilia.

So-called submucous thrush (chronic moniliasis[33, 38] which usually affects adults) is a much deeper seated process than is acute moniliasis (thrush), which is seen especially among children. The former is characterized by slightly adherent, indurated, white to grayish plaques which occur anywhere on the mucous membranes, including the tongue (Fig. 5a). Rockwood and Greenwood reported a fatal case of infection by Monilia albicans, in which there was involvement of the mucous membranes and extensive lesions of the skin. There was no evidence of pulmonary moniliasis, which has a serious prognosis. Submucous thrush usually runs a benign course, but often is intractable to treatment. In many cases it closely simulates the picture of leukoplakia[7] of the mucous membranes. Differentiation may be difficult. Lichen planus can occur on the mucous membranes alone,[12] but usually there are associated lesions elsewhere,

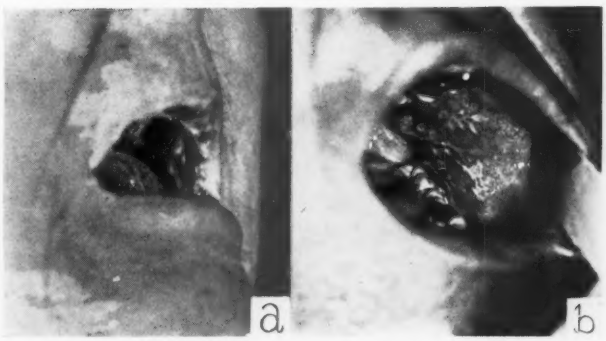

Fig. 5

5a. Submucous thrush of tongue and mucous membranes which lacks the distinct sheen, radiating striæ and discrete papules of lichen planus.

5b. Lichen planus of cheek, showing discrete shiny flat-top polygonal papules, plaques, and striated arrangement.

such as on the wrists and flexor surfaces of the glabrous skin. The lesions of lichen planus are characterized by polygonal, flat-topped, violaceous papules which often merge to form plaques. In the mouth there are also so-called Wickham's striæ, which are accentuations of the normal cleavage lines of the skin (Fig. 5b). Here the lesions are more frequently grayish than they are violaceous in color; often, there are ramifying striæ. No scale can be scraped off. Submucous thrush does not present the striæ. It is somewhat more edematous than lichen planus. The histopathologic picture of lichen planus of the mucous membranes is the same as that seen in the skin, except that at times there is an absence of a granular layer. The histopathologic changes of submucous thrush consist of a more edematous and boggy epidermis, and definite spores and mycelial filaments may be seen even deep in the tissue. Submucous thrush and lichen planus of the oral cavity also must be distinguished from lupus erythematosis of the mucous membranes. In the last condition there usually are associated lesions of lupus erythematosis on the face or elsewhere on the body, which permit a definite clinical differentiation. In lupus erythematosus there frequently is a so-called silvering of the lips as the result of adherent white scaling of both lips. Leukoplakia can be distinguished from these conditions by the absence of the characteristics mentioned for them and by other concomitant findings.[19]

I shall not consider lesions of mucous membrane in systemic diseases such as diabetes, pernicious anemia, purpura, and uremia. I do wish to emphasize that vitamin deficiencies can be a factor in stomatitis and ulcerations of the mouth, for example, in pellagra and scurvy. Pellagra usually presents the syndrome of dermatitis, diarrhea, and dementia; in other words, symptoms referable in varying degree to the cutaneous, gastro-intestinal, and nervous systems. The dermatitis is usually sharply limited to the wrists, like a gauntlet, and to the V of the neck and to the face; in other words, it is limited to the exposed areas. The tongue is swollen, denuded, and sore. Varying degrees of superficial ulceration are seen. The mucous membrane of the mouth is injected in similar fashion. Pellagra may occur secondarily to alcoholism and in association with malignancy or extensive operations of the gastro-intestinal tract which have necessitated a restricted diet.[8]

Mention must be made of acrodynia, which may affect adults. In about two-thirds of the cases there is involvement of the alveoli and buccal mucous membrane. Congestion is present with or without ulceration. There also may be ulcers of the tongue.

Many conditions of the tongue may be associated with variations in acidity of the mouth, or with sensitivity to certain foods, or even with emotional disturbances. This applies especially to so-called burning tongue and transitory benign plaques of the tongue.

Gingivitis and stomatitis may result from the various allergic factors, both through direct contact and true atopic reactions. A complete list of these factors is scarcely feasible. Among the most frequently encountered irritants may be mentioned, lipsticks, toothpastes containing phenolphthalein, and wheat, eggs, milk, chocolate, tomatoes, oranges, cabbage, and even chicken. In the case of foods, when the patient stops eating those to which he is sensitive, he has freedom from attacks. In such cases the stomatitis usually occurs in distinct episodes with free periods intervening. Some persons are hypersensitive to light and have attacks of cheilitis exfoliativa, or eczema of the face. In some cases recurrent attacks of stomatitis, herpes, erosions of the lips, and even vicarious menstruation from the gums[33] are observed during the menstrual cycle. Stomatitis, glossitis, erosions, and even leukoplakia, besides such mild symptoms as metallic taste, salivation, nervous shocks, and burning tongue, also have been attributed to electrogalvanic phenomena set up by dissimilar metallic dentures in the mouth.[17]

Fig. 6

6a. Dermatitis medicamentosa or toxic erythema; involvement of mucous membranes caused by aspirin.

6b. Dermatitis medicamentosa caused by sensitivity to barbital.

Erythema multiforme usually occurs in seasonal attacks in the fall and spring, and is characterized by multiform and "iris lesions" on skin, especially of the extremities, and often by similar lesions or even bullæ on the mucous membranes. When the lesions are extensive, ulcerations, bleeding, and crusting of the mucous membranes result. Many drugs, especially phenolphthalein,[1] can cause an eruption almost duplicating that of erythema multiforme. A person who is sensitive to phenolphthalein may have lesions in the mouth or on the mucous membranes of the genitalia, though, usually, one or more round or oval plaques may be found elsewhere on the glabrous skin. A feature of a phenolphthalein eruption is that it recurs in the same site and results eventually in deeply pigmented, circular spots, which are very disfiguring when they occur on the face of women. Such fixed or transitory drug eruptions may also be caused by the barbiturates, quinine, or asphenamine. Severe stomatitis with ulcerations and bullæ may be the result of the antipyrine-acetanilide group of drugs, the salicylates (Fig. 6a), the bromides, the iodides, all the barbiturates (Fig. 6b), quinine, cinchophen, and so forth.[31 33]

The most important bullous lesion to recognize in the mouth, from the standpoint of the laryngologist, because of its fatal prognosis, is pemphigus.[14 29] This may start in the mouth and throat, and only later on may lesions develop on the body. The primary lesion

Fig. 7. Pemphigus with marked involvement of mucous membranes; one may note discrete noninflammatory primary bullæ on forehead; similar lesions were also seen on mucous membranes.

is a bulla on a noninflammatory base (Fig. 7). The conditions I have just been describing have inflammatory areolæ around the bullous lesions. Patients usually complain of sore throat or mouth and difficulty in swallowing. The mouth, pharynx, and larynx are affected frequently, the esophagus, nasopharynx, and nose very rarely. The bullæ usually appear insidiously and vary in size from a few millimeters to a centimeter or more in diameter. They usually rupture and show secondary inflammatory changes within twenty-four hours. When the disease is severe, there is "beefy-red" ulceration of the soft palate. It is often necessary to observe a patient carefully for several days in order to detect the clear-cut, noninflammatory bulla which represents the primary lesion. Pemphigus is usually fatal, but the type, rapidity, and severity of the process cannot be diagnosed from the appearance of the lesions in the oral cavity.

Pigmentation[24] of the mucous membrane is seen in association with many dermatoses or as the result of treatment. Pigmentation and also gingivitis and stomatitis may result from the use of any of the heavy metals in the treatment of various conditions. Among these metals may be mentioned bismuth, mercury, lead, gold, and silver (argyria). Addison's disease is usually characterized by marked increased pigmentation of the buccal mucosa, which serves as an aid in diagnosis. In acanthosis nigricans, a rare disease of the skin which usually is associated in adults with neoplasm of the abdomen, one frequently sees verrucous lesions of the mouth similar to those occurring on the skin, and occasionally also notes an increase in pigmentation.[21] There may be increased pigmentation of the mucous membranes in hemochromatosis. In this instance, the pigment is the result of deposits of iron and is not the result of melanin.

One can only mention the occasional to frequent involvement of the mucous membranes of the mouth, nose, and pharynx in other dermatoses, such as scleroderma, herpes zoster, impetigo, granulomatous conditions such as yaws, leprosy, leishmaniasis, lymphopathia venereum, granuloma pyogenicum, molluscum contagiosum, and occupational dermotoses,[39] especially those attributable to various metals and chemicals used in industry.[5 6 14 39]

Various dermatologic conditions of the oral, pharyngeal, and laryngeal mucosa have been described. The dermatologist always examines the mouth, nose, and throat carefully for any lesions, as such lesions often are of distinct aid to him in diagnosis of many dermatoses. The otolaryngologist should be able to derive some benefit from looking at the skin of the exposed areas for any evidence of a dermatosis which might aid in the diagnosis of an oral condition. A great many dermatoses are not local conditions limited to the skin and mucous membranes, but are manifestations of systemic disease, and hence call for careful general examination by the internist, and, at times, various laboratory tests should be employed.

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XVII

HEMOLYTIC STREPTOCOCCIC MASTOIDITIS

A Comparative Study of One Hundred Cases in Contagion and in Noncontagion*

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This study began two years ago and was originally planned to be a detailed investigation of hemolytic streptococcic mastoiditis in contagious and noncontagious cases. However, the bacteriology and immunology involved in this study, especially in the contagious group, proved to be of such importance that most of our work and observations have been in this direction.

The hemolytic streptococcus is of particular importance to otologists and well deserves the title, "Public Enemy No. 1." A review of the literature shows it to be the offending organism in from 65 to 88 per cent of mastoid cases reported. Jones gives a very high per cent, namely, 221 out of 252 cultures taken at operation. Dwyer gives 65 per cent. Our Detroit percentage reaches 79, during the March and April season.

Approximately half, or fifty cases, are noncontagious, operated upon in general hospitals. The remainder were operated upon in the Herman Kiefer Hospital, which is a hospital for strictly contagious diseases. All the contagious group was admitted for infectious disease in which mastoiditis developed as a complication. In the noncontagious group the mastoiditis was primary. All the cases studied showed at operation a positive culture of hemolytic streptococci.

The explanation is seen in the increased virulence in the contagious cases; in other words, one out of every four of hemolytic streptococcic mastoiditis was found to be bilateral. This is important to remember, especially when one is confronted with the problem of deciding upon which mastoid to operate. Our rule has been in these bilateral cases, if we have a "one plus" destruction on one side, and a "two plus" destruction on the other side, in the presence of measles

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or scarlet fever, to operate upon both at the same time. A "one plus" destruction, questionable on one side, is much less hazardous in noncontagious cases. Our most important guide has been the rapidity of the invasion, as well as its severity. According to Williams, only 25 per cent of these severe cases, clinically diagnosed as septicemia, show positive blood cultures.

THE AGE INCIDENCE OF HEMOLYTIC STREPTOCOCCIC MASTOIDITIS

Noncontagious			Contagious		
Years	Cases	Per cent	Years	Cases	Per cent
0-5	24	50	0-5	26	54
5-10	11	23	5-10	14	29.5
10-15	1	2	10-15	2	4
15-20	6	12.5	15-20	1	2
20-30	2	4.	20-30	3	6.5
30-40	3	6.5	30-40	1	2
40-50	1	2	40-50	0	0
			50-60	0	0
			60-70	1	2
		48		48	100

UNILATERAL AND BILATERAL INVOLVEMENT

Noncontagious		Contagious	
Unilateral	72%	Unilateral	76%
Bilateral	28%	Bilateral	24%

BLOOD STUDIES

Noncontagious		Contagious	
White blood count	14,328	White blood count	16,445
Polymorphonuclear	70%	Polymorphonuclear	77%

HEMOGLOBIN AND RED BLOOD COUNTS IN CONTAGION

Average hemoglobin	69%	Red blood cells	3,800,000

There was nothing unusual in the blood findings. In one case of measles a leucopenia with 2,800 white blood cells was found, in the presence of a "three plus" destruction. The white blood count continued postoperatively in over 90 per cent of our cases, in both groups, and was felt to be a toxic reaction. It was not uncommon to find a decrease of 10 per cent or more in the hemoglobin in a two day period. This is not at all unusual in the septic or toxic cases.

X-ray Studies

DESTRUCTIVE CHANGES IN MASTOIDITIS

	X-ray	At Operation
Contagious	2.5+	3+
Noncontagious	2+	2+

This finding is clinically wrong, yet correct from the record of these cases. Our x-ray studies were graded in one, two, three or four plus destruction. (One plus—approximately 25 per cent destruction.) From an x-ray standpoint a much more accurate record of destruction was noted in the noncontagious cases. In a study of a very large series of mastoid plates, in contagious cases, especially in scarlet fever, clear detail is rare, as these plates are usually very hazy or foggy. The more severe the toxemia, the more milky white and more undependable the radiograph. This was especially true in the severe toxic cases of scarlet fever and measles. Organisms other than the hemolytic streptococcus do not, as a rule, give us as milky a radiograph.

Our records agree with those of Allison, namely, if a culture from the aural discharge is pure staphylococcus, in contradistinction to the hemolytic streptococcus, we may temporize. If the pneumococcus is found, one has to be on the lookout for any early meningeal signs. If hemolytic streptococci are found in the aural discharge, in the presence of contagion, prompt action is usually necessary, even in spite of a questionabie x-ray finding.

Page[1] reports that, in 300 cases, there were three times as many mastoid operations required in the cases of spontaneous drum rupture, as in those in which paracentesis was done. Our records show very little, if any, delay in this destructive process, even with early well-performed paracentesis. We recognize two distinct types of acute inflammation of ear drums in these hemolytic streptococcus infected ears, in scarlet fever. Early these drums are flat and deep red, often with very little pain, and if incised, only a serosanguinous fluid will be found. On the following day many of them will be paler, especially in the center, with some bulging. If incised at this time the fluid will still be serous and blood tinged but seldom purulent. Lewis verifies Page's findings. In our records spontaneous rupture, as against paracentesis, gave us a ratio of two to one. Records and statistics most certainly uphold us in advising an early paracentesis. In our hospital records we can show a very high percentage of cases, especially in the moderate and severe scarlet fever and measles, in which, in the short space of twenty-four hours, mastoid destruction extended from one or two plus destruction to even a three and sometimes four plus destruction at operation.

STANDARD GROUPING ALL TYPES OF SCARLET FEVER TABLE

Mild temperature	101 or less
Moderate	101—102.5
Severe	102.5—higher

KIEFER GROUPING

Scarlet Fever	1934-35-36	Scarlet Fever	1934-35-36
Moderate	56%	Septic	3%
Severe	36%	Toxic	1%
Following surgery	4%		

The septic group included those of the dirty or purulent type of nose, the invasion of the ears and of the cervical glands, with chills, and the picket fence temperature chart. These are the few cases in which immunotransfusion gave the best results.

In the toxic cases there was a very severe rash, a fast pulse and concommitently a toxic heart, with a sustained elevated temperature; these were the only cases in which we used convalescent human serum intravenously.

INCIDENCE—COMPLICATIONS—SCARLET FEVER

Antitoxin or convalescent serum given on:

	132 Cases 1st and 2nd Days	97 Cases 3rd and 4th Days	22 Cases After 4th Day
Adenitis	3.7%	7.2%	4.5%
Otitis media	6.1%	8.2%	18.1%
Mastoiditis		1.03%	13.6%
Nephritis		1.03%	4.5%
Sinusitis		2.0%	

If serum was given on the first or second day of the disease the secondary complications were limited to adenitis and otitis media; if delayed, a sharp rise in both the secondary complications and the severity was noted.

As to the percentage of loss of hearing, our records are very unreliable and do not permit publication. Few of our cases of toxic measles, during the 1934 outbreak, showed a greater loss than similar scarlet fever cases. We are unable to explain this.

Kopetzky[4] feels that the early acute scarlet fever mastoid is of the acute necrotic type and tends to remain as a chronic purulent otitis media, with cholesteatoma, and with its usual hearing loss. Kopetzky also states that mastoiditis, developing later in the course of scarlet fever, is of less severity, not requiring immediate paracentesis. This is contrary to our records and findings.

BACTERIOLOGY

For the purpose of this paper, we are assuming that the hemolytic streptococcus designated by many as the streptococci scarletinæ are the cause of this type of mastoiditis and also of scarlet fever. It is also found in many skin, peripheral, peritoneal, lung infections and in pneumonia.

Some difference of opinion prevails among bacteriologists and immunologists in reference to this. Gabritschewsky believes that hemolytic streptococcus lives in the tonsils and the nasopharynx, and the exotoxins from these produce the rash and invasive complications. Gordon and others believe it to be a virus disease.

SUMMARY OF 297 CULTURES

Positive for hemolytic streptococci.

	Number	Per cent	
Spontaneous	183	61.61	
No definite type	53	17.84	
Type I	11	3.70	
Type II	25	8.41	
Type III	7	2.35	20.53%
Type IV	14	4.71	
Types II and IV (mixed)	4	1.34	
Total	297	99.96	

SUMMARY OF CULTURES FROM 1,587 SWABS

	Number	Per cent
No growth	48	3.02
Negative for hemolytic streptococcus	1,233	77.69
Mixed growth	9	.56
Hemolytic streptococcus	297	18.71
Total	1,587	99.98

This is a table of 1,587 cultures of cases admitted during 1934 and 1935. The 18 per cent positive streptococcus hemolyticus is very low in comparison to the usual findings reported in the literature, or around 50 per cent more when carefully taken by physicians. This error is emphasized by the fact that 22 per cent of the discharge cultures of the throat, out of some 23,000 cases, showed streptococcus hemolyticus. Our rule has been to require two negative cultures for hemolytic streptococci from the ear, nose or throat before discharging the patient. The Board of Health of Detroit is hopeful of arriving at a standardized method of labeling those carriers.

The result of this research work, on typing the hemolytic streptococci, was done at the Herman Kiefer Hospital and revealed six

different groups of agglutination, in what was morphologically the same organism.

This is the result of our series only. Park and Williams mention only four groups of agglutination of scarlet fever and three groups in erysipelas. Also, in his series, the hemolytic streptococcus cross-agglutinates some of the scarlet fever and erysipelas strains, respectively. These observers believe that hemolysis is produced very early in the life of the hemolytic streptococci and also that the secondary complications are due to complex exotoxins.

The controversy that exists among research workers is outside of the realm of otology. Some observers, notably Tunnicliff,[7] have recognized as many as thirty-six different types of hemolytic streptococci, based on the varying agglutinizing and opsonizing properties. The agglutinizing identification may lead us to a more select and more rational serum therapy. Comparable to our present typing and grouping of the pneumococcus, this problem is, of course, one of bacteriology, but well deserves the co-operation and encouragement of the otologists and pediatricians.

In recent years many authorities have published their results with immunotransfusion and antitoxin therapy, including both horse serum and convalescent human serum. Lyons[8] states: "Hemolytic streptococci produced disease by virtue of two attributes, namely, the ability of a bacterium to invade the body tissues and the capacity to form toxins. These are separate qualities, requiring separate analyses for the effective neutralization within the body." The immunologic evidence to support this conception is now sufficiently complete to warrant the continued therapeutic use of this specific antitoxin.

Both Lyons and Williams[3] express the view that while the antitoxin will combat the toxemia reactions, it will have no effect upon the invasive power of the bacteria. Our findings, however, are more hopeful as we have reduced complications. The many arguments, still in progress, as to the specificity of the exo- and endotoxin of the hemolytic streptococcus still do not hinder our present use of pooled convalescent serum.

Lyons has shown that polyvalent antistreptococcic serums lack specific opsonizing antibodies for a large number of strains. Our findings coincide with Baum's[9]—that is, that convalescent serum contains anti-bodies which may not be bacteriologically specific, but therapeutically specific against streptococcic infection, which may clinically be a different disease from that from which the donor has recovered. Baum also has selected the agglutinative method of typing as

the simplest and the quickest. As otologists, I believe it is necessary that we should have a working clinical understanding of this agglutinative process.

To emphasize this, I would like to report two cases of mastoiditis in children in whom cultures showed hemolytic streptococcus; both had uncomplicated mastoidectomies. They were noncontagious cases, with no dural or sinus exposure in either. The postoperative temperature varied from 101 to 102½ and there was cervical adenitis. Both cases improved at once, after administration of 30 cc. of convalescent human serum. In another boy, aged 14, with uncomplicated hemolytic streptococcus infection (noncontagious), a mastoidectomy was performed. Severe septicemia immediately followed, with a negative blood culture. We were unable to get an immuno-donor for transfusion and instead gave three intramuscular injections of 30 cc. of convalescent human serum, without shock and with very pleasing results. In all, we have had only five cases in this noncontagious hemolytic streptococcic group, and are aware that the number is entirely too small to justify any more than the expression that our results, so far, have been so pleasing that we are still hopeful and intend to continue.

Sewall,[5] Baum and others have had comparable results in like cases. The use of convalescent human serum is justifiable in these noncontagious cases, especially those of either the septic or toxic type.

Several requests for the details of the preparation, manner of administering, and results of the use of convalescent human serum, have prompted their inclusion in this report.

Donors of fifteen years or over, weight 100 pounds or over, are selected from thirty days to six weeks after recovery from scarlet fever. The blood is typed, Kahn tested, the red, white count and the hemoglobin recorded. The donors appear at 4:00 P. M., having had no lunch; 250 to 300 cc. of blood are withdrawn, of which only half is eventually available as serum. To date, this serum has been kept in sealed ampules; however, with the instillation of a new desiccating machine, it will be distributed in the dry form in ampoules, similar to salvarsan, requiring for use only the addition of sterile water. It is claimed that by this process there will be no loss of potency and that it may be kept for years.

One point worthy of mention is if the convalescent donor is sensitive to horse serum, this sensitivity may pass on to one likewise sensitive. Two cases in our series developed a moderate urticaria in one week. Only one fatality, in a large number of cases, and that one in

a severe toxic scarlet fever, followed immediately the intravenous administration of convalescent serum.

Our method of obtaining this serum is original with the local Board of Health, who were able to compensate each donor, or mother, with a $10.00 fee. The amounts and supply have been scant or below the demand. Both the attending otologists and pediatricians can co-operate with the bacteriologist by collecting serum from their convalescent patients. There is, I believe, a much greater use for this serum than we have heretofore recognized, and there will be plenty available at all times if we will but extend our efforts in collecting it.

In severe toxic cases the convalescent serum may be given intravenously. It is, however, in our hospital a method of last resort, since we believe it is more hazardous.

The injection is made over the fascia lata, on the lateral side of the thigh, at the junction of the lower two-thirds and the upper one-third, with the needle pointing down. The advantages of this over gluteal injection are that the reaction is controllable by tourniquet above the injection site; it is less painful to a patient lying in bed; there are no important structures liable to trauma, and there is quicker absorption from the fascia lata than from the glutens muscle. In our last 1,000 injections by this tourniquet method not one reaction was encountered.

This type of antibacterial serum has not had widespread use, due to the difficulty of obtaining it, especially in the smaller cities and the rural communities, to the failure to use it early, and to the great tendency to employ it only as a last resort.

Most observers agree as to the clinical action, namely, that no improvement is seen in the first twelve hours. It is fair in the second twelve hours and most marked in the third twelve hours.

Our results, in a very large number of cases, have been more pleasing than in years past when using commercial horse serum. This has a much greater potency; it is more expensive, produces more shock in the patient and lastly, the most important, there is ever present the fear of serum sickness. Cases receiving 40 to 60 cc. of horse serum will remember the serum sickness long after the disease is forgotten.

Park[10] states that convalescent human serum confers immunity that lasts longer. In our series of cases both sera acted similarly, in that they caused the rash to fade and lowered the temperature.

In some of the cases, where neither convalescent human serum nor horse serum was available, we did what we thought was next

best, namely, injected 60 cc. of the parent's whole blood, to supply nutrition and antibodies. It is my belief that in selected noncontagious cases of hemolytic streptococcic mastoiditis, convalescent human serum will prove to be of value and an admirable supplement to our present day surgery and management of these cases. Our motto in our contagious hospital's use of convalescent serum has been: Serum early and repeat. Benefits outweigh dangers.

This also may apply in a modified degree to our future management of hemolytic streptococcic mastoiditis in noncontagious cases.

SUMMARY

1. There is a very high incidence of hemolytic streptococcus found in mastoiditis in both contagious and noncontagious cases.

2. There is an increased virulence and invasiveness of hemolytic streptococci in contagious cases.

3. The mastoid radiograph in the contagious cases is unreliable.

4. Convalescent human serum is preferable to horse serum antitoxin if administered early and repeated.

5. Fascia lata injections are preferable to gluteal injections.

6. Convalescent human serum is believed to be of distinct value in hemolytic streptococcic mastoiditis, and worthy of trial in the noncontagious cases, with septicemia.

910 HOFMAN BLDG.

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XVIII

A NEW METHOD OF RHINOPLASTY FOR THE SINKING OF THE TIP OF THE NOSE*

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Montreal

All surgeons who devote themselves to the esthetics of the nose admit that it is very difficult to correct a sinking of the tip of the nose. The methods described offer a certain improvement but are not entirely satisfactory. Recently I have had recourse to a new process, which is embodied in the following history:

REPORT OF A CASE

L. S., a male, 22 years of age, sought my advice, on September 23, 1935, about a depression of the end of the nose. He said that he never received any traumatism and that his deformity was congenital.

The tip of the nose was particularly soft and deeply sunken. The nostrils were slightly dilated. The nasal bones were in a normal state. In short, the malformation was localized solely in the inferior region of the cartilaginous septum.

Anterior rhinoscopy showed a well defined straight septum, without thickening. The turbinated bones had not increased in size.

There was nothing of note in the rhinopharynx and in the ears.

I proposed a rhinoplasty. An antiseptic nasal salve was prescribed, and half an hour before the intervention I injected a solution of hyoscine, morphin and cactoid.

Having irrigated the nasal fossæ, I made a local injection of novocain-adrenalin. Under the control of the index finger, placed in the right nostril, I incised the left throughout the length of the mucocutaneous junction. Great care was exercised in order to avoid any perforation. Having elevated all the neighboring tissue, I dug a tunnel over the tip of the nose to the nasal bones. Still under local anesthesia, I took a long piece of cartilage from the first floating rib on the right. Stripped of its perichondrium, it was separated in two, and the thinnest part, after being modeled, was placed in the nasal vestibular wound. This graft, in direct contact with the quadrilateral cartilage, had its point of support on the superior maxilla, and pushing up the tip of the nose. Silk and very thin needles were used to

*Read before the Premier Congrès Européen de Chirurgie Structive, Bruxelles, 3-4, Oct., 1936.

suture the wound. In order to increase the esthetic effect and at the same time to protect the tip of the nose against pressure caused by the extremity of the cartilaginous beam, I made a small external vertical incision on the skin which covers the columella, with the object of rejoining the tunnel already dug. This communication established, the second piece of cartilage, the ticker, was then cut lengthwise in two and, after modeling, one part was introduced by this opening as far as the nasal bones. Finally, the second half was placed at the tip of the nose in order to cover the extremity of the graft. I then sutured the cutaneous incision with silk, by means of small conjunctival needles, and a light compressive dressing of the two nostrils was applied. This wound was protected with a piece of iodoform gauze maintained by two bands of adhesive plaster. According to my habit, I applied finally, under the pyramid, a splint of copper, retained by adhesive tape. Three layers of sutures closed the costal wound, and an ordinary abdominal dressing was applied.

The dressing of the nostrils was changed after two days, and then renewed every twenty-four hours. From the seventh day I began to remove the stitches of the skin covering the columella and those of the vestibule. The sutures of the costal wound were allowed to remain a little longer. Sixteen days after the intervention the patient left the hospital absolutely cured. At this time the tip of the nose was well remounted, and was to be found in direct line with the nasal bones. The nostrils had a normal opening. Eight months after the operation the patient wrote me that he was always satisfied and that his nose was perfectly straight.*

If we examine the different methods used to correct a sinking of the tip of the nose, we see that each is suited to a certain type of deformity. In the presence of a nose too long and slightly flat at its point, we must, by the endonasal route, separate the columella from the septum, resect small slices of quadrilateral cartilage, separate the skin of the nasal pyramid, and suture on each side the vestibular mucous membrane, after having removed the exuberant part. In order permanently to stabilize this skin and by the same fact lift up the tip of the nose we must apply bands of adhesive plaster with traction, at first vertical, and afterwards lateral.

If the sinking manifests itself on a soft nose, with nostrils sufficiently large, Joseph of Berlin advises the entire detachment of the columella from the septum, by means of an external incision, to uncover the two alar cartilages, and then to suture them in order to bring them nearer and to lift up the tip of the nose. In certain cases

*I saw this patient on January 11, 1937. The cure is permanent.

this method can give fair enough results, but generally the cicatricial tissue, which forms after the operation, shrinks with time, changing the appearance. The operation can also be performed by the endonasal route, but it is easy to see that this is much more laborious and more uncertain.

Sheehan of New York makes an incision of the skin which covers the columella, and tunnels the tip of the nose. Having taken a piece of costal cartilage, he models it in such a way that two parts, about equal and bent at a right angle, are retained by the perichondrium. This graft is then placed at the tip of the nose as far as the nasal bones, and in the wound of the columella. In hands as skillful and experienced as his, it is possible to obtain excellent results. However, we must admit that for a good correction the graft must be perfectly prepared from all points of view, and that the mucous membrane of the nasal vestibule on each side must not be damaged.

Moulonguet of Paris corrected a very marked sinking of the tip of the nose, following a traumatism dating back to early infancy, where there was no cartilage of the septum and of the columella, in the following manner. Under anesthesia with chloroform, a small transverse incision of one-half centimeter was made just below the end of the nose, and a subcutaneous canal of four centimeters was elevated as far as the nasal bones. A second incision started from the middle part of the first one, transversely, and bisecting the columella and the septum to a depth of one and one-half centimeters. He took a piece of cartilage from the edge of the ninth rib, stripped of its perichondrium, presenting more or less the form of a square whose longest branch was two and one-half centimeters. This graft once placed has its point in contact with the nasal bones, its inferior part with the nasal spine, and its superior portion pushing up the tip of the nose. The author of this process reports having obtained a very good profile. As to the front view, the graft took a slight curve to the right, which forced him later to paraffin to correct this concavity. Possibly Dr. Moulonguet's patient requested the chloroform. It would have been much easier to perform it, without pain, under local anesthesia, which would have allowed him to distend the tissues, up to a certain point, and especially those of the nasal septum which, according to his description, were reduced to "a simple mucous fiber." I do not understand the need of a transverse incision, of one-half centimeter in length, below the tip of the nose, before making a tunnel as far as the nasal bones, nor the necessity of making a subcutanous canal of four centimeters as far as these same bones, when it was necessary to put in a cartilaginous graft of but two and one-half centimeters.

For this particular case the result has been very good. However, I doubt that this method could be applied in a practical manner for the correction of a sinking tip of the nose in general, because there would always be the danger of a perforation of the mucous membrane of the nose at the time of the bisection of the columella and of the septum. We all know that this little accident might have, as a consequence, an infection at first, and then the elimination of the graft. I shall not dwell upon the cutaneous cicatrix which with time almost entirely disappears.

The setting of a cartilaginous stay, to push up the tip of the nose, has already been described, but this simple method is insufficient to give to the pyramid a straight line. Moreover, it causes the end of the nose to become more or less nippled at the place where the pressure is found. The creation, from the front to the rear, of a tunnel in the columella, after an incision from five mm. to one cm., recommended by certain authors, is not practical, because this tunnel is too small to receive a graft strong enough definitely to push forward the tip of the nose, and besides its application presents the danger of rupture during severe exercise. With the piece of cartilage which I introduce by the external route as far as the nasal bones, I can obtain at will the desired correction. Finally, the last graft, placed in contact with the preceding one, has for its object not only to increase the effect of the nasal correction, but also to protect the tissues in covering the cartilaginous beam at its lobular extremity.

It is very important to place the index finger in the right nostril during the section, from the front to the rear of the mucous membrane, which separates the columella from the septum to the left, in order to exercise a control and consequently to prevent the perforation. So as to give more extension to this mucous membrane incised, with the object of introducing a cartilaginous stay as large as possible, absolutely straight and free from its perichondrium, we must have recourse to a dissection of its borders. The graft is then placed in contact with the septum, and it is easy to protect it perfectly in suturing with silk, by means of very fine needles, the vestibular wound. Finally there remains but to practice a small vertical incision below the tip of the nose, to communicate the opening with the tunnel already created at the time of the section of the mucous membrane separating the septum from the columella, and to place therein the two pieces of cartilage.

For all rhinoplasties dealing with a pronounced lesion, where it is necessary to have recourse to a graft, I employ the cartilage—as

I have already stated in a previous paper—and in preference the cartilage of the first floating rib.[1] Anatomically this adapts itself particularly well to the modeling, and this graft alone is ample for the correction of all deformities produced by an insufficiency of the nasal skeleton. Moreover, this cartilage can be taken without pain, under local anesthesia, and the postoperative sequelæ are always very simple.

In a nose flattened at its end, widened and stretched, we must at first bring together the alæ, according to the methods well described by the authors, before lifting up the tip of the nose with a cartilaginous beam, and then applying, by the external route, the two other grafts.

I have described in this paper the method that I recommend for the correction of the sinking of the tip of the nose, and underlined its superiority over all others. This operation is particularly easy, is practiced without any pain under local anesthesia, and assures the best possible guarantees as to the esthetic results. I regret to be unable to furnish any illustration, because my patient refused to be photographed either before or after the operation.

3434 Rue St. Hubert.

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XIX

TRIFACIAL NEURALGIA

JOHN H. CHILDREY, M.D.

SAN FRANCISCO

Neuralgia of the fifth nerve, trifacial or trigeminal neuralgia or tic douloureux, is a much dreaded disease which does not clear up spontaneously. Nothing definite is known of its etiology. It has been suggested plausibly that the disease is the result of previous infection of the Gasserian ganglion or of infection elsewhere in the body, although removal of foci of infection affords no relief.

Temporary relief from pain is possible only by means of alcohol injections or by avulsion of the peripheral nerve. Any other period of relief noted after other types of treatment can be explained readily as a period of quiescence common to the disease, for the pain invariably returns with its usual regularity.

Permanent relief from the pain follows division of the sensory root of the ganglion above the crest of the petrous bone. In any case, it is advisable first to perform from one to three alcohol injections before advising the radical operation. This permits the patient to experience temporarily the very annoying numbness of the face which will accompany the relief of pain. He then knows what to expect should he decide later to have the nerve divided. After one or two recurrences of the neuralgia, patients gladly choose the numbness instead of the excruciating pain. The alcohol injection is indicated particularly for patients in whom the condition has not been long present and for those who, because of advanced age, cardiac or renal disease, or for other causes, are bad risks. Many of these patients are over 60 years of age. Trifacial neuralgia is uncommon in young individuals. The patients usually visit a number of doctors seeking relief and give a history of having had bad teeth extracted or sinuses operated on without benefit. The average period of relief following alcohol injection is seven months. Occasionally, it may be as long as two and a half years.

The right side is probably more often affected, and the second and third more than the first division of the nerve. At first only one branch of the fifth nerve is affected, but as the disease progresses other branches are involved. The pain is brought on by external irri-

Fig. 1

tation, such as washing the face, brushing the teeth, chewing, swallowing, talking or exposure to air currents. The pain, present only during waking hours, is best described as similar to an electric shock through the face. It may be described as sudden, sharp, stabbing or shooting pain, often excruciating in severity. It involves the area supplied by the branch or branches of the affected nerve and lasts only a few seconds to a minute or two. The patient may go into all sorts of contortions in an effort to obtain relief. He may chew, or bend over, hold or rub the face or even fall to the floor during a paroxysm. One elderly woman under our observation screamed at the top of her voice during attacks. The disease incapacitates the patient and may lead to permanent invalidism or mental disorders.

There are trigger areas associated with the onset of the attacks, and these spots are situated usually, but not always, in the area of the branch affected. The upper or lower lip, side of the nostril, gum, or floor of the mouth are common situations of the trigger spot. The patient is careful to avoid irritating these. The paroxysm may occur several or many times in a day, or there may be periods of several weeks of relative freedom from pain.

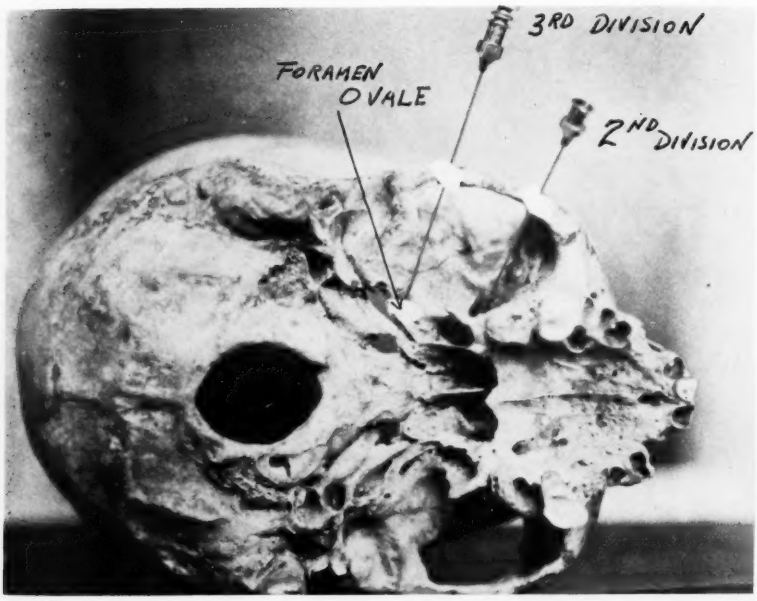

Fig. 2

We prefer to inject the alcohol under local anesthesia. Although this takes a little longer, one is more certain of getting the alcohol in the proper place. A hypodermic needle is used for the first division, which is injected at the supra-orbital notch. The second division is injected at the foramen rotundum, a three-inch spinal puncture needle being introduced at the lower border of the zygoma, just behind the malar prominence. It enters in a slightly upward, backward direction on the plane of the outer canthus of the opposite eye, to a depth of 6 to 7 cm., depending on the individual. The third division is reached at the foramen ovale; with the patient's mouth slightly open, the spinal needle is inserted one inch anterior to the tragus and at the lower border of the zygoma, at right angles to the plane of the zygoma and almost horizontally inward to a depth of 4.5 to 5 cm., when it impinges upon the margin of the foramen ovale. With the needle in place, the trocar is withdrawn, a syringe attached and aspiration attempted, thus avoiding injection into a vessel. Then a few cc. of 1 per cent novocain are injected and after a few minutes, as a rule, the patient states that numbness of the area supplied by that branch of the nerve has occurred. Then 1½ cc. 95 per cent alcohol

is injected, the trocar reinserted, and the needle withdrawn. The pain caused by the alcohol is controlled by sedatives, and is of relatively short duration.

The man pictured, aged 71, had had a cataract successfully removed from the left eye in 1934. On his second admission, in September, 1936, he complained of severe shooting pain in the right lower jaw, of six months' duration, and increasing in severity. Our diagnosis was trifacial neuralgia of the third division on the right side. The trigger area was on the right lower alveolus. The injection of alcohol at the foramen ovale resulted in relief from the pain, and this relief has continued to the present time. The mouth is opened slightly during the injection so that the needle will not encounter the lower jaw. (Figs. 1 and 2.)

GREENS' EYE HOSPITAL.

XX

A CASE OF BENIGN GIANT CELL TUMOR OF THE ETHMOID LABYRINTH WITH A REVIEW OF THE LITERATURE*

MERRILL WATTLES, M.D.

BOSTON

From the time of Ambroise Paré there have been innumerable cases of benign giant cell tumors reported in the literature, but after a careful review of the cases reported from 1860 through 1935, it was found that there have been only seven cases of this type of tumor reported that originated in the ethmoid labyrinth. For this reason it was thought the finding of this rather common tumor in this unusual location warranted reporting.

REPORT OF A CASE

The patient, a white male, 21 years old, was admitted to the Out Patient Department of the Massachusetts Eye and Ear Infirmary on June 6, 1936. He was first seen in the Eye Department on that date. Examination revealed his vision to be 20/20 in each eye with a slight blurring in the left eye.

Following x-ray examination the patient was admitted to the Infirmary on June 9, 1936, with a diagnosis of mucocele of the left posterior ethmoid cells. His chief complaint was a protrusion of the left eye and headache. His family history was entirely negative except that a sister died of cancer. He had measles, mumps, chickenpox, scarlet fever, pneumonia and diphtheria in childhood. There were no nose and throat complaints up to the present illness, which began six months prior to admission, at which time he was struck in the left eye. No disability was noted following this accident, but the tissues about the eye remained discolored for two to three weeks. During this period a friend told him that his left eye was more prominent than his right. From that time he noted that the prominence of the left eye gradually increased and that it protruded outward and downward. Accompanying the eye signs, the patient had frontal headache, which gradually increased in severity up to the time of admission. It was constant and had become almost unbearable during the past month. Intermittent diplopia was first noted six weeks prior to admission.

The general physical examination was entirely negative. The left eye was protruding downward and outward. The pupils were regular, round, equal and reacted to light and accommodation. The scleræ were clear. The examination of

*From the Mosher Laboratory, Massachusetts Eye and Ear Infirmary.

The author wishes to express his appreciation to Dr. Philip E. Meltzer, who treated the case in the Infirmary, for permission to report it.

the nose revealed a congested, reddened and slightly swollen mucous membrane. No abnormal secretions or polypi were present. There was a slight high deviation of the nasal septum to the right. The remainder of the nose and throat was negative.

The routine examinations of the blood and urine were negative. The Hinton test was reported negative.

On June 10, 1936, an external exenteration of the left ethmoid labyrinth was done under ether anesthesia by Dr. Meltzer and Dr. Irgens. On reflecting the periosteum over the anterior portion of the orbital plate of the ethmoid bone, granulation tissue was found which had broken through from the ethmoid cells into the orbit. Posterior to this the bone was of the thickness of an egg shell, and superiorly the expanding tumor had encroached on the floor of the anterior cranial fossa, where on removing the polypoid mucous membrane of the posterior ethmoid cells the dura was exposed for an area 2 cm. in diameter. The dura was normal in appearance and intact. The patient was returned to the ward in good condition.

The tissue, which was examined in the Mosher Laboratory, consisted of several portions of the tumor from the region of the posterior ethmoid cells and from the anterior ethmoid cells over the left orbit.

Gross examination of the specimens revealed several pieces of tissue which were congested and hemorrhagic. No capsule was noted. The cut section showed numerous cystic areas filled with a sanguineous fluid throughout the tissue, and in many places the section had both the appearance and the consistency of healthy granulations. There was no bone found.

Microscopic sections showed the predominating cells of three types, namely, spindle cells, round cells and giant cells. The spindle cells made up the bulk of the tissue, which according to most authors, is indicative of a more or less quiescent state in the growth of the tumor. Another striking feature of the tissue was its marked vascularity. There were numerous new capillary vessels with a proliferation of the endothelial cells. There was much hemorrhage throughout the tissue and very typical large cystlike spaces containing both normal and broken down red blood cells. A third important process was noted in many areas formed principally by the spindle cells. Embedded in the stroma of the spindle cells were found many small spicules of new formed bone. These varied somewhat in age, but it could be seen that they were all in the process of formation, and not one showed any sign of destruction. This was recognized by the healthy appearance of the bone and the fact that on all sides the spicules were surrounded by osteoblasts. This type of formation is very characteristic of this tumor, but is found in only about one case in ten, according to Mallory. The most important finding on which the diagnosis rested was the presence of innumerable giant cells scattered throughout the tissue. They were found in the greatest numbers at the periphery. They were very large, with a rather clear cytoplasm taking the basic stain and containing many large regular darkly staining nuclei grouped in the center of the cell. No mitotic figures appeared in any of the sections. Had these been present, it could have been said that this specimen was a classical example of the disease under discussion.

From the patient's history, physical examination, and both the gross and microscopic examination of the tumor, it was thought the most probable diagnosis was

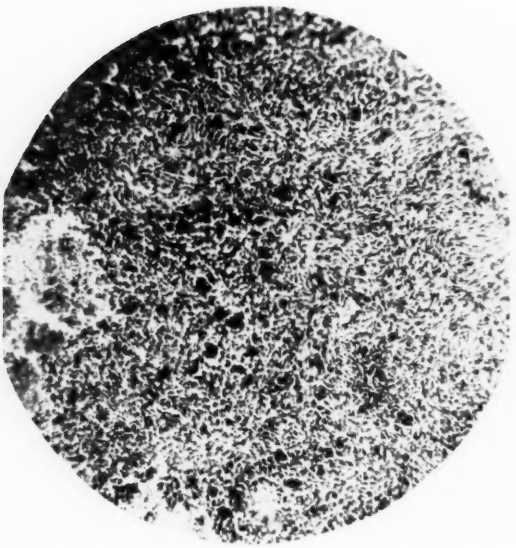

Fig. 1. A low power photomicrograph of a portion of the tumor near the periphery. Note the large number of giant cells, the scarcity of round cells and the predominance of the spindle cells.

benign giant cell tumor of the ethmoid labyrinth. This diagnosis was confirmed by Drs. Mallory and Castleman of the Department of Pathology, Massachusetts General Hospital.

Following the operation, the patient's condition remained satisfactory, and all the packing was removed by the sixth postoperative day. On the seventh day examination by Dr. Moore of the Eye Department revealed the following: Left eye markedly proptosed. Lids edematous. Ocular movements restricted in all fields. Media clear. Fundus normal. Exophthalmometer reading, right eye, 13 mm., and left eye 23 mm. Exophthalmos of 10 mm. on the left. The patient complained of burning of the left eye during the examination with the ophthalmoscope. The diagnosis was retrobulbar edema or tumor. Examination nine days postoperative revealed a clean, healthy healing wound, but it was noted that there was still a marked proptosis of the left eye. Dr. A. S. MacMillan re-examined the patient by x-ray on June 23, 1936, and reported a slight thickening of the mucous membrane of the left antrum, a postoperative defect of the left ethmoid with no sign of remaining tumor, and the remaining sinuses negative. On June 24, 1936, Dr. Moore again examined the patient and reported a proptosis of the left eye of 7 mm. as compared with 10 mm. of eight days previously.

The following day Dr. E. W. Herman of the Tumor Clinic saw the patient in consultation and advised further observation for one month. The patient was discharged June 26, 1936, to return to the Out Patient Department weekly for

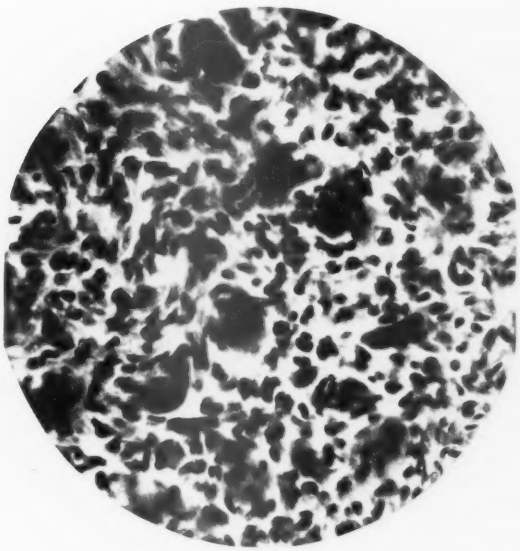

Fig. 2. A high power photomicrograph showing fourteen giant cells. These cells contain ten to thirty dark nuclei arranged at the center of the cell. The predominating cells are the spindle cell type, but an occasional round cell is noted.

observation. At the time of this report the left eye was still proptosed 7 mm. The eye movements, however, were good in all fields. On July 24, 1936, the patient was re-examined by x-ray and Dr. MacMillan reported a postoperative left ethmoid wound with part of the lower rim of the ethmoid labyrinth remaining posteriorly, but no sign of a tumor mass. The remaining sinuses were clear.

REVIEW OF THE LITERATURE

Ewing[1] defines the giant cell tumor as a low grade neoplastic process, usually single, sometimes multiple, affecting mainly the epiphysis of the long bones, but appearing in almost any bone of the body, producing multicystic excavation with a widening of the shaft and pursuing a prolonged and generally progressive course, but failing to yield metastases.

As mentioned at the beginning, this type of tumor has been known since the time of Paré,[1] who reported several cases of the maxilla. In 1845, Lebert[2] differentiated the carcinoma and the sarcomas of bone, but thought that the giant cell tumor as we know it today should be classified as a sarcoma. Following Lebert, Paget,[2] in

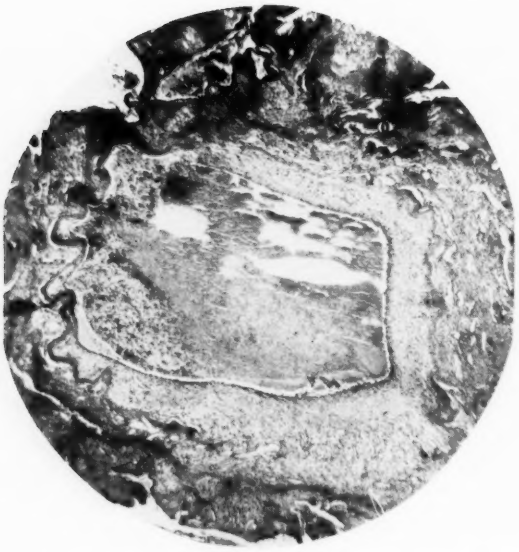

Fig. 3. A low power photomicrograph of a large section of the tumor showing a very large cyst in the center filled with both normal and broken down red blood cells. To the right of the cyst are seen numerous small, newly formed capillaries. These findings are characteristic and account for the great vascularity of the giant-cell tumor.

1853, first recognized the giant cells in this tumor, but attached no particular significance to them. The classical description, including clinical, gross and microscopic pathology, was recorded first by Nélaton[3] in 1860. His report was so complete that there has been little added since its publication. Nineteen years later Gross[2] discussed the benign character of the giant cell tumors of bone, but stated that he was not sure that the tumor might not become malignant. Many of the early authors considered this tumor benign, but in America it was on the whole thought to be malignant until Bloodgood,[4 19] in 1910, called attention to the fact that it was a benign tumor and suggested that it be called "benign giant cell tumor." Since this time there has been a great deal of discussion as to whether it might not in some cases become malignant.

The benign giant cell tumor occurs in both males and females of practically all age groups. Most authors agree, however, that it is far more common in the female and the time of greatest incidence is

Fig. 4. A low power photomicrograph showing an area in which there is marked hemorrhage throughout the tissue. Areas such as this and also those shown in Fig. 3 were found in abundance throughout the entire tumor.

during the second and third decades. It has been stated above that this tumor may involve almost any bone in the body. We shall see later why it cannot involve all the bones, and particularly all the bones of the skull. The bones which are affected, and the order in which they are most frequently involved, are as follows: ([1 5 6 7 9 10 11 12 14 15].) Long bones, small bones, maxilla, mandible, vertebrae, ribs, scapula, clavicle, ethmoid, sphenoid, temporal and frontal bone. There is also a case reported by Weider[13] of giant cell tumor of the cartilaginous portion of the nasal septum. In the case of involvement of the frontal bone, the writer doubts that the diagnosis was correct.

The etiology of the giant cell tumor and the cells from which it receives its name have been the subject of much discussion and study since they were first described. Simmons[10] states, in his review of giant cell cases, that 64 per cent of all the patients gave a history of injury prior to the complaint of subjective symptoms. Likewise Geschickter and Copeland,[7] in a review of 226 cases of this tumor, obtained a history of trauma in from 40 to 50 per cent of all cases. Ewing,[1] Linde[2] and many other authors agree that trauma may be

Fig. 5. A low power photomicrograph showing new bone formation. Note the osteoblasts along the edge of the spicules of bone and the large size of the cells within the bone. There is no sign of bone destruction, and there are no giant cells present.

an etiologic factor, but they conclude that it is doubtful that trauma plays a very important part in normal bone. Another school of thought sponsored by Mallory,[17] Codman,[11] Lubarsch,[11] Haythorn[18] and Konjetzney[11] hold the tumor is actually an inflammatory process, the giant cells being merely foreign body cells, such as one might expect to find in almost any chronic inflammation. Guenther[4] thought the principal causal factor was a disturbance in the secretion or a disturbance in the metabolism of the secretion of the parathyroid glands. Johnson[16] decided that too many theories of no value had been advanced up to 1930, so after examination of eleven cases of benign giant cell tumor he concluded the giant cells were definitely formed from the endothelial cells lining the capillaries in this very vascular tissue. He reports that giant cells were found along the capillary walls replacing the endothelium in some locations, and "vacuoles" seen in the giant cells were fine capillaries which had pierced the cell and formed new vessels. As far as the writer knows, this observation has not been confirmed by anyone else.

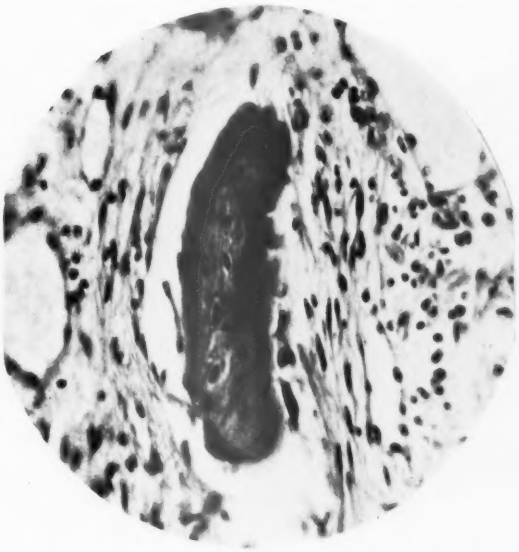

Fig. 6. A high power photomicrograph of the new bone formation seen in Fig. 5. Osteoblasts are seen around the edge of the spicule and the large size of the new cells within the bone is noted.

The theory which seems most logical and the one which leaves less doubt in the reader's mind as to the etiology of the tumor and the one which explains the very striking fact that it does not occur in all the bones of the body was that advanced by Geschickter and Copeland.[6 7 9] Granting that the giant cell tumor was more common in the third decade and that the site of the tumor was usually at the epiphysis, they made an elaborate study of the embryology and the histogenesis of both the long bones and those of the skull so as to take into consideration bone preformed both from cartilage and membrane. Their studies revealed the following points: Ossification occurs late in the epiphyseal portion of the bone, and this accounts for the rather late occurrence of the tumor, also at the epiphyseal end the periosteum is less active and the thin cortex allows the tumor to progress. It was also noted that this tumor occurs at a time when the marked reparative powers of bone have been lost. The authors found that at one point in the process of the formation of bone from cartilage there occurred a concentration of mesenchymal cells, in which the nuclei became dark and finally an agglutination of the cells took

place to form giant cells or osteoblasts. These cells perforated the perichondral bone and after them came the new capillaries and other elements. When this process was at its height, they stated, it had many of the appearances of a giant cell tumor. Their conclusion was that giant cell tumor was the result of abnormal hyperplasia of the osteoclasts taking place in the histogenesis of intracartilaginous bone. These facts were further substantiated by a study of giant cell tumors of the face and skull, for it has long been known that certain of these bones have their origin in cartilage and others in membrane. If their theory was correct, the bones formed from membrane should be immune to this particular tumor. Thus, in a study of twenty-two cases of giant cell tumor of the head, excluding epulis of the alveolar margin, they found two cases from the great wing of the sphenoid, six cases from the ethmoid and fourteen cases from the mandible. In all these cases it was found that the tumors originated from the parts of these bones which were formed from cartilage. They concluded that giant cell tumors can only be found in those parts of the skull which are entirely cartilage in origin and the fact that they arose from the centers of ossification of these cartilages proved their theory that the origin of the giant cell tumor was correct.

There has been much written concerning the benign nature of this tumor. Nélaton,[3] in his original article, stated that this was probably a benign tumor. Bloodgood[19] was the first author in this country to emphasize its benign character. Working with Bloodgood, Geschickter and Copeland,[8] in a review of a series of cases, concluded that the tumor is not malignant but may recur, depending on the treatment given. Pierce,[11] after a review of the literature, states that the tumor is considered benign by Paget, Gray, Virchow, Gross, Codman and Kolody. Boyd[12] goes so far as to say that if the tumor were found to be malignant the original diagnosis was incorrect or a sarcomatous change had been superimposed on the original tumor. Other authors believe, however, as expressed by Ewing,[1] that after long periods of growth and complications the giant cell tumor may become metastatic and cause death. Sharing this belief were Chatterton and Flagstad,[20] who had a case which was malignant clinically and by x-ray, but a biopsy showed it to be benign. More radical views were held by Simmons,[10] who after finding six deaths of metastasis out of eighty-two cases, concluded that malignant change did take place in the giant cell tumor. Coley[26] goes so far as to state that about 15 per cent of the cases become malignant and many of these he thought were due to poor treatment. The predominance of opinion favored the benign view, but the writers who differed agreed that the matter was not closed.

Simmons,[10] in his review, reports that there have been at least twenty forms of treatment recommended. The ones most used and the results are as follows:

Form of Treatment	Percentage of Cures
Amputation	100 per cent
Complete excision	100 per cent
Curettement with cautery	60 per cent
Curettement, cautery and x-ray	72 per cent
Radiation	75 per cent
Coley's toxins	42 per cent

Where there are so many different forms of treatment for the same condition, there are likewise as many authors who think their method is the one of choice. Among those who believe surgery offers the best results are Linde,[2] Geschickter and Copeland,[9] Coley,[26] Cotton,[21] Platt[22] and Bloodgood.[19] Most of these writers are of the opinion that, secondary to surgery, irradiation has a place in the treatment. Those who feel that x-ray offers more satisfactory treatment in most cases are Herendeen,[25] Pfahler and Parry,[24] Evans and Leuctia,[23] and lastly, Ewing,[1] who thinks that x-ray irradiation is rapidly replacing surgery. Here again, where there seems to be such a great difference in opinion, there is still much more evidence needed before any one method can be chosen as a specific remedy.

SUMMARY AND CONCLUSION

1. This is a report of a case of benign giant cell tumor of the ethmoid labyrinth.

2. In a review of the literature from 1860 through 1935, only seven cases of benign giant cell tumor of the ethmoid labyrinth were found reported.

3. A review of the literature is presented, including the history, incidence, etiology, malignancy and treatment of benign giant cell tumor.

4. Benign giant cell tumor of the ethmoid labyrinth is a rare disease. The question of its benign character, its treatment and possibly the question of its etiology should be the subject of further study.

243 CHARLES ST.

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XXI

PREVENTION OF DEAFNESS IN THE SCHOOL CHILD*

BURT R. SHURLY, M.D.

DETROIT

It has been said of old that an ounce of prevention is worth a pound of cure. It is time today that the public at large, and the medical profession in their organizations, have realized that preventive medicine, as a whole, or as specialized otolaryngology, must devote more of time, money and research to this field, which offers so much for practical accomplishment. If 3,000,000 or more children of pre-school or school age call for better scientific aid and attention along these lines of prevention I am sure the League for the Hard of Hearing, the profession and the otologists are ready to study and classify the special problems in the prevention of deafness.

A grand start has been made and the papers of Doctors Fowler, Coates, Shambaugh, Phillips and others have done much to stir enthusiasm and bring results.

The school child offers a practical opportunity to apply our methods of prevention.

Some of the plans successfully carried out by the National Tuberculosis Association should be used more extensively. The Christmas Seal has provided funds that have promoted national organization and led to an actual prevention of tuberculosis and the saving of many lives.

The prevention of deafness, under the auspices of the League for the Hard of Hearing and the medical profession, needs more funds, extensive organization, public and medical education and general propaganda. The facts of prevention must be advertised by those who know how to conduct the campaign successfully. The medical profession cannot advertise, but they can talk over the radio, in the schools, all over the world.

The parent-teacher associations, the teachers and the pupils must know the facts of prevention of deafness as they do know them about

*This is an address broadcast during the convention of the American Academy of Ophthalmology and Otolaryngology, in New York, September 26th to October 3rd, 1936. This talk was prepared to interest and benefit the laity and is printed here chiefly as an example which may stimulate other similar appeals.

tuberculosis. In Detroit, where we have 400,000 public and parochial school children, we have established a card system in the office of each principal, whereon is registered the name of the family doctor employed by the parents.

The importance of early diagnosis and the practical steps to be taken may be presented to the principal or teacher by the family doctor. Where a school doctor or nurse is in attendance a follow-up contact with the family physician and parents becomes readily possible, and reference to the otologist, clinic, deaf school, or hospital may be attained with greater speed and intelligence. There is a woeful lack of understanding among parents, teachers and some family physicians of the dangers of the acute and suppurating ear. Education and more of it in the schools is our preventive remedy. With the name and address of the family doctor on the card of every school child in the U. S. A., it is only a step further to record on the same card the name of the otolaryngologist or doctor who removed the tonsils and adenoids, or who cared for the ears of the child when there was an early loss of hearing.

New York, with Dr. Fowler and a splendid corps of otologists, has led the way in hearing tests for school children. This propaganda and scientific work must be extended throughout the country to solve the problem of early diagnosis. Hearing saving classes are of like importance with sight saving classes, and the establishment of more schools for the deaf in the larger educational systems may well be advocated. The interest of school boards throughout the land should be aroused to effect appropriation of funds for beginning work in the amelioration of deafness. These may be approached by the medical profession when more funds for education are available. If we are to prevent deafness the fight must carry on by virtue of scientific facts in relation thereto. We know that deafmutism is either congenital, sporadic or acquired. The congenital variety is a problem of eugenics. Deafmutes propagate deafmutes, and if we will refine our laws and arouse public opinion on intermarriage, demanding standards at least equal to, if not better, than the ordinary rules of animal husbandry, congenital deafmutism could be eliminated. The sporadic case is pathologic and may happen to the best family blood. Prophylaxis and better treatment of syphilis is rapidly diminishing the number of cases of deafness from that cause.

Otosclerosis, degenerative disease of the nerve of hearing and its bony envelopes, has a strong family tendency to repeat, and in the light of our present knowledge can be remedied only by respect for the laws of eugenics.

It is estimated that acquired deafness in childhood is, in 10.6 per cent of the cases, the result of disease of the nervous system, such as meningitis for the most part; 28.3 per cent from infectious diseases, and 61.15 per cent from ear disease and injury of primary origin. Here scarlet fever, measles, influenza, whooping cough and diphtheria take their toll. Diphtheria, as a cause, is rapidly fading under the splendid preventive measure of toxin-antitoxin. Scarlet fever, because of convalescent serum, earlier diagnosis, and isolation, is diminishing in virulence, and resultant damage to the ears is also decreasing. Influenza may be a causative factor in many ear lesions. The school medical service must therefore become more alert to middle ear infections causing 61 per cent of the deafness in childhood.

We have unfortunately no specific for the prevention of the type of deafness which attends suppurative disease. We have no antitoxin like those for diphtheria, or tetanus; or specific drugs like quinine for malaria. We can cause suppuration in the middle ear of the white rat or monkey with deficient vitamin A feeding. We can prevent scurvy with vitamin C and beriberi with vitamin B and rickets with vitamin D.

The 60 per cent of deafness caused by pus germs must be prevented, in the light of present knowledge, with all these adjuncts that are known to raise our resistance to infection. There are those whose immunity is so great that neither colds nor suppurative disease invade their system. The secret has not been unlocked; but the management of suppuration in the nose, throat and ear may turn the tide toward recovery if early diagnosis, early opening of the ear drum and early draining of sinus infection are done in acute stages of infection. What not to do requires exercise of common sense, good judgment, a study of the individual and an estimate of the virulence of the infection, balanced by experience and a prompt choice of medical and surgical therapy, applied without delay. Science and research must unfold more and more the mysteries of biochemistry and selective action of the body cells. The art and the science of medicine must work together in the foreground of prompt remedial treatments. The role of the lymphatics cannot be forgotten in the battle to put down acute suppurative disease, that may later produce deafness. We know, but we sometimes forget, the splendid action of calomel and magnesia in our early cases.

If we are to recognize that we of the medical profession are dealing with an economic, sociologic and public health problem, we must co-ordinate our forces under the banner of the League for the Hard of Hearing and collectively adopt a plan of procedure and

propaganda that will apply our scientific knowledge along lines that will be acceptable to the school authorities of the several states.

We must have money for nation-wide testing of hearing among school children.

The degree of handicap must be known and a definite classification made and recorded. It will then be possible to outline and apply such successful remedies as may be available at the time. Research and the progress of scientific treatment will open up new and startling discoveries as time goes on. When we have a card index of every school child; when his family physician and his otologist are noted; when classes in lip reading and special classes with vocational instructions for the various degrees of the hard of hearing are available in all the schools, certainly greater opportunities for our handicapped will open in various fields of endeavor.

The following points might well be kept in mind:

1. The danger of the acute ear infection must be emphasized.

2. Tonsils and adenoids, when harmful, must be completely removed, and especially the lymphoid tissue in the fossæ of Rosenmuller overlying the eustachian tubes.

3. Acute coryza or the common cold must be given better treatment.

4. The problems of immunity must be studied.

5. The questions of diet, with milk, eggs, leafy vegetables and a good supply of mineral salts, must be considered.

6. Hygiene, with proper common sense as to clothing and ventilation for the individual in and out of school, should be applied.

7. Ears should be protected in swimming pools, and those with damaged ears prohibited the use of pools.

8. Swimming pools with imperfect disinfection should be closed.

9. All suppurating ears should be under medical examination, advice and treatment.

10. Periodic hearing tests are necessary for those with loss of hearing. The group audiometer is best for our school children.

11. Sinus infection and nasal obstruction should receive all necessary and beneficial operations and treatment.

12. The excessive use of alcohol, tobacco, quinine and salicylates, especially among pregnant women, should be discouraged.

13. The ever increasing noises of a great city, the use of firearms and cannon and any sounds that fatigue the nerve mechanism have an influence of some importance in bringing about deafness.

Time marches on, and neither science nor the earth stands still. A nation-wide campaign to educate and interest school authorities, who have responsibilities of administration, should be conducted on a larger scale. We need the services of a great advertising expert, who will definitely map out the campaign.

First, may we find out who are heard of hearing; and then, what is the remedy for those who can be benefited? And next: Can we lead the way to better hearing where aid is possible?

Although complete prevention of deafness cannot be attained until science has mastered the problem of the common cold, still, a vast concentration of thought on this subject by the united forces at work can produce marvelous results.

Let us join hands with the League for the Hard of Hearing in a greater effort to educate the people and the professions in the best known scientific facts toward the prevention of deafness.

He that hath ears to hear, let him hear.

62 Adams Ave. W.

XXII

FRACTURES OF THE FACIAL BONES

WILLIAM D. GILL, M.D.

SAN ANTONIO

Fractures of the facial bones have assumed an increasingly important place in surgery within recent years due principally to the greatly increased number of automobile and airplane accidents. Formerly such fractures were often referred to as bicycle injuries, but more recently have been designated as steering post and guest passenger injuries because of their frequent association with automobile accidents. There is not a medical man so remotely removed from the highways of civilization that he may not be suddenly and unexpectedly called upon to treat such injuries, although they fall more particularly within the province of the surgeon practicing ophthalmology and otolaryngology because of the usual coincident involvement of the structures within the orbit or of the nasal accessory sinuses.

Straith[1] reminds us that the "guest passenger" in an automobile is often more seriously injured than the driver, due to the fact that the latter can usually brace himself against the steering wheel when an impact is anticipated. The degree of injury encountered ranges from minor fractures without displacement of fragments to extensive crushing injuries in which surgical shock is an invariable accompaniment. There is always some degree of associated injury of the soft parts, ranging from slight contusions to lacerated wounds and actual loss of tissue.

Emphysema, involving the soft parts, is prone to occur in certain types of fractures of the facial bones, especially those involving the anterior wall of the antrum or medial wall of the orbit. In fractures involving the frontal sinus, emphysema is relatively infrequent but does occur.

In fractures involving the bones forming the orbit, there may be sufficient displacement of fragments to seriously compromise the eyeball, impinging upon or perforating it and causing serious impairment of its motility. This is especially likely to occur in transverse fractures of the orbit, or when the malar bone has been dislocated upward and inward.

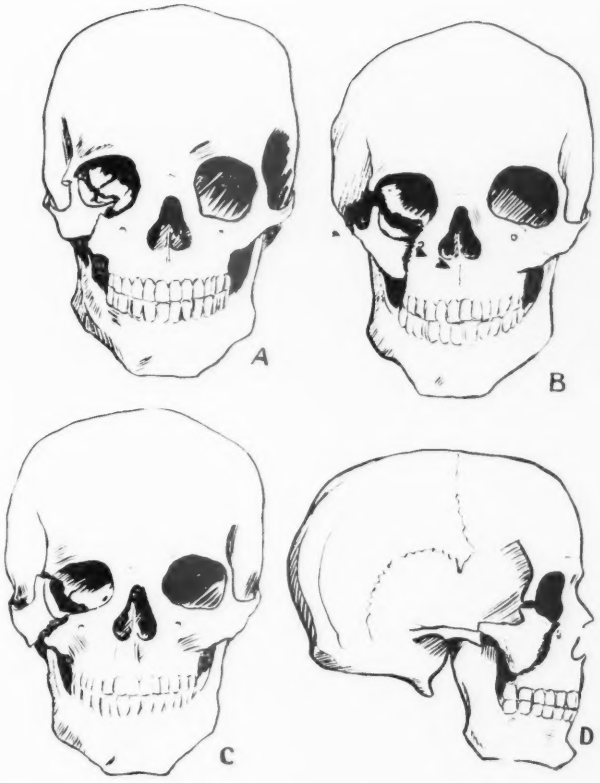

Fig. 1. Diagrammatic representation of several types of fractures involving the malar bone.

A. Inward and upward displacement of malar bone.

B. Downward and inward displacement of malar bone.

C. Outward displacement of malar bone.

D. Lateral representation of skull showing manner in which the temporal process of the malar bone may impinge on the mandible, interfering with its motion.

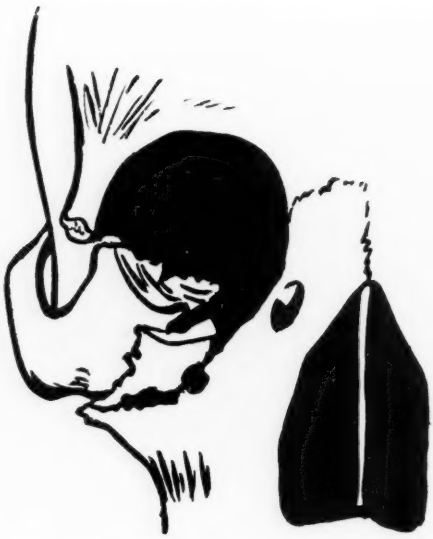

Fig. 2. The inconstant infra-orbital suture may be responsible for a separate small fragment of bone being detached in fractures involving the antral walls.

The malar bone, next to the nasal bones, is the facial bone most frequently fractured, and for this reason the antrum of Highmore is often involved. An occasional instance is encountered in which the superior maxilla is fractured in extraction of teeth, and the adjacent maxillary sinus inadvertently opened. The presence of dentigerous cysts or large granulomas due to infection about the apices of the teeth increases the probability of such fractures as rarefaction and absorption of bone takes place during their formation.

Some degree of hemorrhage into the soft parts is always present; it may be slight and concealed, involving only the soft parts, or it may be alarming in degree, often filling the adjacent nasal accessory sinuses and calling for radical measures to effect its control. In compound fractures this complication may be especially troublesome.

It is well to bear in mind that extensive intraorbital hemorrhage may cause optic atrophy through sustained pressure on the optic nerve and that hemorrhage and emphysema may occur coincidentally, although the latter complication is usually of minor significance, as it rapidly subsides.

Fig. 3. Inspection of the upper teeth is imperative in fractures involving the upper jaw. Note the medial displacement of the teeth on the upper right side in this patient who has suffered a fracture involving the right superior maxilla and malar bone.

The frontal, maxillary, ethmoid and sphenoid sinuses may be involved through the action of direct violence, the radiation of fracture lines, or through the penetration of sharp objects. The writer has encountered fractures involving the paranasal sinuses which were caused by the penetration of missiles, such as shotgun pellets, portions of firearms, pieces of wood, as well as a variety of other agents too numerous to mention.

The danger of meningitis is always present in fractures of the facial bones, especially when the nasal accessory sinuses are involved, and is greatly increased when infection is present within the sinuses.

Suppurative osteitis is an occasional sequel to fractures of the facial bones, and is usually traceable to pre-existing infection in the paranasal sinuses, or to gross contamination of comminuted fractures at the time of injury.

The presence of fractures of the facial bones should be suspected in all injuries of the face due to direct violence, and should be either eliminated or verified by inspection, palpation and x-ray examination,

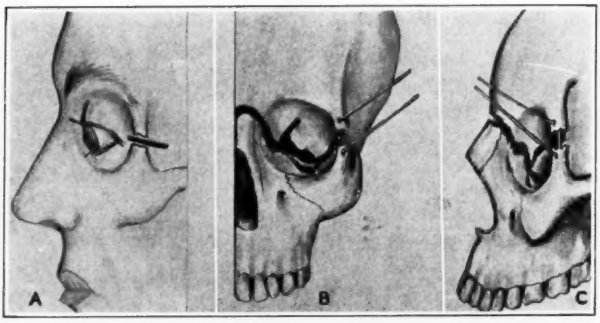

Fig. 4. Method of wiring facial bones to the skull in transverse fractures passing through both orbits.

(a) Skin incision: Care must be exercised to avoid injuring fibers of the facial nerve, orbicularis oculi muscle, or the external palpebral ligament.

(b) Silver wire passed through drill holes in the frontal process of the zygoma and the zygomatic process of the frontal. A blunt instrument is inserted between the eyeball and the eyelids at the outer canthus to prevent injury to the eyeball from the drill, or in passing the wire suture.

(c) Same as (b), viewed from the side. There is less danger of opening the cranial cavity in drilling the frontal process, if done in this manner.

as a slight and apparently insignificant facial injury is often associated with a fracture involving the underlying bone structure.

In the treatment of facial bone fractures, it is advisable that reduction be effected as promptly as possible, barring the presence of extensive hemorrhage, surgical shock or other traumatism, such as fractures of the bones of the skull and visceral injury, which will naturally receive primary consideration. It is well to bear in mind that delay in reduction beyond a reasonable length of time will result in disappointment, due to the fact that interfragmentary clots become firmly organized, or that beginning callus formation interferes with reposition because of the presence of relatively unyielding tissue between the fragments. Swelling of overlying tissues may occasion delay in reduction for several days because one cannot accurately determine by palpation when reposition has been effected.

There are numerous methods of effecting reduction of displaced fragments, all of which possess special points of merit. In malar bone fractures, Matas[2] pulls the displaced fragment into position by encircling it with a silver wire; Codman[3] and Kazanjiian[4] use bullet forceps, while Wurdemann[5] employs a strong resection hook. In frac-

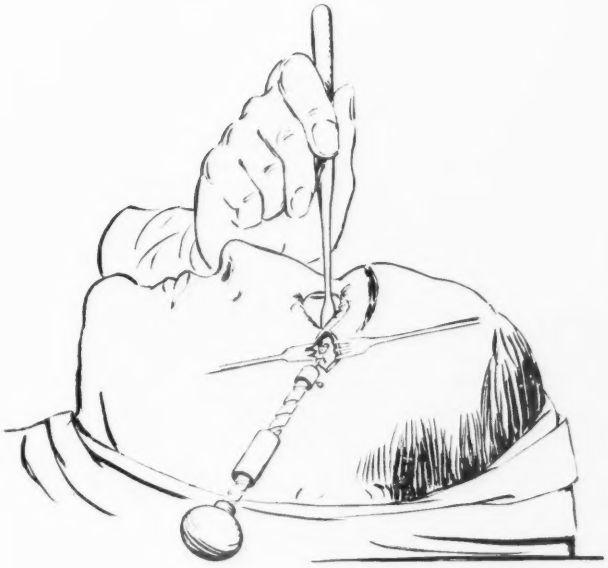

Fig. 5. Method used to protect the eyeball from injury in drilling the zygomatic process of the frontal bone in preparation for wiring of fractures in this region.

tures of the malar bone, and in crushing injuries involving the superior maxilla, Lothrop[6] and Naftzger[7] open the antrum in a manner similar to that employed in radical sinus surgery. Shea[8] employs a Ritter sound introduced through a naso-antral window to push the displaced fragments into position. Evans[9] advocates the latter method and in addition points out that the procedure provides nasal drainage for the involved antrum. Goldthwaite[10] elevates a depressed zygomatic arch by means of a flat periosteal elevator introduced through a buccal incision above the last upper molar tooth. Roberts[11] uses a screw device which is twisted into the displaced fragment through a stab incision in the soft parts. The writer[12] uses a special forceps, similar to a large towel forceps, to grasp the displaced fragment through the skin and move it into position. In the absence of this instrument an ordinary towel forceps may be utilized, although it is not usually heavy enough for the strenuous service it will be called upon to perform. The latter method leaves no more scarring than would be caused by the puncture of a hypodermic needle, and is easily accomplished under interfragmentary local anesthesia with 2 per cent novo-

Fig. 6. Roentgenogram showing fracture through both orbits, which has been wired according to the method described in the text. Silver wire has been passed through drill holes in the frontal process of the zygoma and zygomatic process of the frontal bone, and lower fragments pulled into position by this means.

cain solution. Massive infiltration with anesthetic solutions should be avoided, as palpation, which is an essential guide in reduction, will be interfered with.

Retaining appliances are not usually required in fractures of slight degree, as the interlocking of serrated edges is usually sufficient for fixation. In extensive injuries interdental splints or wiring of fragments may be necessary. There is one particular type of fracture in which great difficulty is usually encountered in obtaining fixation; that is, the transverse type of fracture extending through the orbits and across the bridge of the nose, in which the bones of the face are separated from the base of the skull. Oftentimes attempts are made to obtain fixation in such fractures by the use of plaster of paris head caps and interdental appliances which not infrequently lead to many trials and eventual disappointment to both the patient and the surgeon. Occasionally one will encounter a patient sufficiently stolid to endure the discomfort of the plaster cap fixation, but the use of such an appliance is not at all conducive to the feeling of good will which should exist between the patient and the surgeon. In my expe-

rience fixation with head bands and leather straps has proved to be worse than useless. This difficulty is easily overcome by wiring through the zygomatic process of the frontal, and the frontal process of the zygoma or malar bone,[13,14] on each side, pulling the displaced fragments into apposition. Nature has provided this support for fixation, which may be easily and satisfactorily utilized with minimum discomfort to the injured patient.

Blair,[15] Bodine[16] and Kazanjiian[17] emphasize in their writings that in certain badly comminuted fractures of the upper jaw interdental appliances are oftentimes of great value.

Fractures of the malar bone, in which the zygomatic arch is angulated outward, may be reduced satisfactorily by delivering a series of blows on the angulated bone with a mallet through a padded block of wood held in apposition with the skin. Supplementary traction with a seizing forceps applied anteriorly may be of considerable assistance in effecting reduction.

It is advisable to use some type of antiseptic within the nasal fossæ in fractures involving the sinuses in order to diminish the possibility of infection extending into the surrounding tissues.

Adhesions between the septum and lateral walls of the nose should be guarded against as they readily occur when there is marked swelling of the nasal mucous membrane.

Expert dental assistance is essential in injuries associated with malocclusion of the teeth or where the coronoid process of the mandible has been impinged upon by displaced fragments.

It may be necessary to await the subsidence of edema before attempting reduction of fractures of the facial bones because of the necessity of employing palpation in effecting reduction. After swelling has masked the deformity it is best to delay reduction until it has subsided, as palpation, which is a necessary guide in reduction, is interfered with. Reduction can usually be effected within a week or ten days after receipt of injury, as swelling of the soft parts will usually subside within that length of time.

A detailed discussion of fractures of the nasal bones and of the lower jaw has been purposely omitted from this article, as they constitute special subjects which have been adequately covered elsewhere. The frontal bone has been included because of its frequent involvement in facial injuries, although it is not anatomically classified as one of the facial bones.

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Clinical Notes and New Instruments

XXIII

JACKSTONES IN THE UPPER FOOD PASSAGES: REPORTS WITH ESOPHAGOSCOPIC OBSERVATIONS ON ELEVEN CASES*

GABRIEL TUCKER, M.D.

PHILADELPHIA

In a series of 475 foreign bodies in the upper food passages, pharynx, esophagus and stomach, there were eleven cases of jackstone. The foreign bodies were found to be in the pharynx, esophagus, cervical esophagus, thoracic esophagus and one in the stomach at the time of examination of the patients. Investigation of jackstone cases reported shows no case in the larynx or tracheobronchial tree. The jackstone, because of its peculiar shape, has a tendency to embed itself in the tissues at its point of lodgment in the esophagus particularly, and this makes it a very dangerous foreign body and one that is very difficult of removal without trauma. Examination of jackstones on sale at a toy store revealed that four sizes are obtainable, but the tendency at present seemed to be to limit the sale to the medium size. Inspection of the various sizes lead at once to the conclusion that the greatest factor in trauma from the jackstone as a foreign body is the relative size of the jackstone and the lumen of the food passage in which the foreign body is lodged.

In the one case of an adult in which the jackstone was found in the stomach the uninterrupted passage out of the stomach and through the gastro-intestinal tract would seem to indicate that if the foreign body passed spontaneously into the stomach there would probably be no difficulty in its passage through the intestinal tract. In ten of the eleven patients in which the jackstone lodged in the esophagus or pharynx the ages varied from twenty-one months to six years. Death occurred in one patient, twenty-two months of age, from ill advised attempts at removal with a bristle's probang before admission. In another child, twenty-one months of age, death proba-

*Presented before the meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, Newark, N. J., January 3, 1936.

bly occurred from lung abscess which developed after two unsuccessful attempts at removal of the foreign body before the patient was admitted to the Bronchoscopic Clinic, the abscess being present on admission. In this case the parents signed a release after the jackstone had been removed, and the esophageal condition had cleared up; follow-up report was not obtained.

REPORT OF CASES

CASE 1 (Fbdy. No. 1647T)—A boy, three years of age, choked when an older child placed a jackstone in his mouth. The foreign body passed into the cervical esophagus where roentgen-ray examination revealed it lodged thirty hours after the accident, when the child was admitted to the hospital. Removal of the foreign body was accomplished by the dangling method. Without anesthesia an esophageal speculum was introduced, the globular end of an upward projecting prong was grasped with a ball forceps and the jackstone was dislodged by gentle traction, allowing it to dangle just beyond the tip of the speculum as the speculum and foreign body were withdrawn.

Result: No complications. Recovery uneventful.

CASE 2 (Fbdy. No. 1699T)—A girl, twenty-two months of age, choked while playing on the floor with a jackstone. She was taken immediately to a physician who attempted unsuccessfully to remove the foreign body with a bristle's probang. The child developed marked dyspnea and dysphagia and was brought to the Bronchoscopic Clinic within twelve hours after the accident. On admission the child was severely dyspneic and had a temperature of 104. X-ray examination showed marked emphysema in the neck and the jackstone lodged in the upper cervical esophagus. Because of the urgent dyspnea the larynx was examined shortly after admission. Without anesthesia the larynx was lifted forward with a child-size laryngeal speculum and a deep laceration was revealed in the postpharyngeal wall. The jackstone could be clearly seen back of the cricoid, the globular tip of an upward projecting prong was grasped with cupped forceps and the foreign body was easily dislodged and removed by gentle traction. Dyspnea was not relieved by the removal of the foreign body and tracheotomy was done, which relieved entirely the dyspnea. The child died, however, at the end of twenty-four hours from an extension of the mediastinal emphysema and infection.

Comment: Ill advised attempts at removal produced the complications in this case.

CASE 3 (Fbdy. No. 1730T)—A girl, six years of age, choked while playing with jackstones. She was brought to the Bronchoscopic Clinic within four hours of the accident. X-ray examination showed the jackstone to be in the esophagus at the level of the suprasternal notch. There was no dyspnea, the child complaining of slight difficulty in swallowing. There had been no previous attempts at removal. The larynx was lifted forward with an adolescent laryngoscope and the usual technic of removal by the dangling method was carried out without anesthesia. There were no complications and convalescence was uneventful.

CASE 4 (Fbdy. No. 1961T)—A colored boy, six years of age, came to the out-patient department complaining of soreness and stiffness of his neck and some difficulty in swallowing. He had been treated by a private physician for a period of several weeks for a sore throat. Examination at the hospital showed a swelling on the left side of his neck and marked tenderness over the area between the cricoid and the left sternoclavicular joint. He could swallow liquids only and these with great difficulty. His temperature was 101 and there was marked dyspnea on slight exertion. Roentgen examination showed a large jackstone in the esophagus at the level of the sixth cervical vertebræ. The history was obtained that the boy had choked on a jackstone fifty-two days before admission. The child was referred to the Bronchoscopic Clinic, where examination showed him to be urgently dyspneic and with the evidence of severe infection in the neck. A low tracheotomy was done immediately. The dyspnea was entirely relieved and the child was able to swallow liquids with much less difficulty. The neck infection drained partially through the tracheotomy wound which was left open, and on the third day following tracheotomy the inflammatory reaction had subsided somewhat and it was considered safe to attempt removal of the foreign body. Without anesthesia a 9 mm. by 30 cm. full lumen esophagoscope was introduced, the jackstone presented, and when it was loosened from its bed in the esophageal wall a large quantity of pus was liberated and aspirated through the esophagoscope. Biplane fluoroscope guidance was used to keep the behavior of the foreign body under observation during withdrawal. The large size of the esophagoscope was sufficient to open the upper esophagus so that the foreign body could be removed through the cricopharyngeus without trauma during withdrawal. After the foreign body was removed the tracheal compression and dyspnea were relieved. The infection in the neck drained into the esophagus. The convalescence, although protracted, was uneventful, and the patient was decannulated without difficulty.

Fig. 1. Case 4. Photograph of boy after esophagoscopic removal of a jackstone which had remained in the esophagus for 52 days. A scar at the suprasternal notch shows the position of the preliminary tracheotomy before removal. Child was decannulated without difficulty.

Comment:

1. It is remarkable that this child had a jackstone in the esophagus without the medical attendants discovering it for fifty-two days. The long sojourn resulted in peri-esophageal abscess due to a gradual ulceration of the foreign body through the esophageal wall.

2. Preliminary tracheotomy made removal through the mouth and evacuation of the abscess without further mediastinal trauma possible.

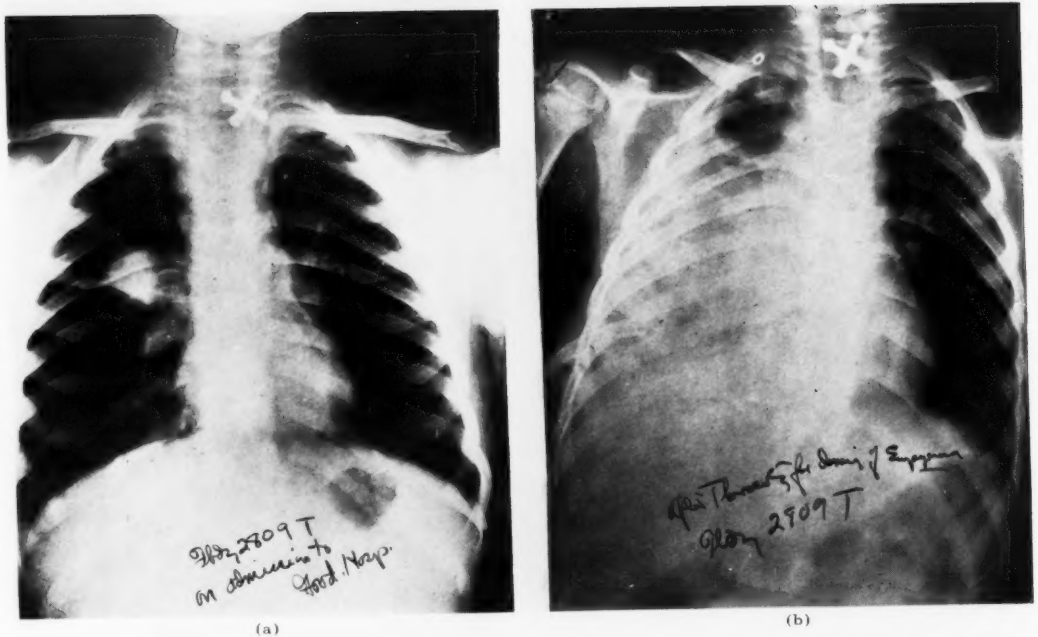
CASE 5 (Fbdy. No. 2269T)—A girl, twenty-one months of age, choked while running with a jackstone in her mouth. The foreign body passed into the upper esophagus, where x-ray examination at a local hospital, where she was admitted, revealed it. Two un-

successful attempts at removal of the jackstone with the administration of a general anesthesia at each attempt was made. Thirty-six days after the jackstone had entered the esophagus the child was referred to the Bronchoscopic Clinic at the Graduate Hospital of the University of Pennsylvania, where she was admitted. The child was somewhat dyspneic and had considerable difficulty in swallowing. X-ray examination showed the foreign body in the upper esophagus with evidence of periesophageal infection. There was also evidence of abscess of the right lung. The child was kept under observation for a few days and the jackstone was removed without anesthesia by the dangling method, using a 6 full lumen esophagoscope. The esophageal condition cleared up satisfactorily within ten days. The pulmonary condition, however, persisted and the parents signed a release and removed the child from the hospital. The child's condition was quite serious, and a follow-up report was not obtained. It is probable that the child succumbed to the pulmonary abscess.
Comment:

1. The small size of the child and the relatively large size of the jackstone made the removal extremely difficult.

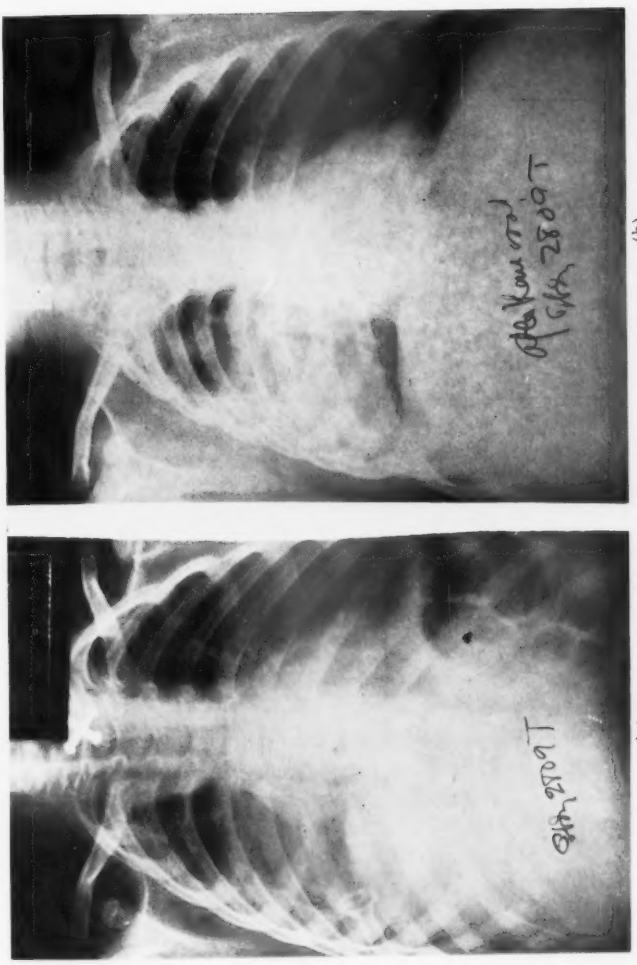
CASE 6 (Fbdy. No. 2634)—A boy, three years of age, choked while playing with a jackstone. He was taken immediately to the hospital, where an x-ray examination showed it to be lodged in the esophagus at the suprasternal notch. The foreign body was removed within twenty-four hours after the accident. The jackstone was presented with an adolescent laryngoscope, and cupped forceps were used to grasp the globular end on one of the upward projecting prongs. The jackstone was removed by the dangling method. There were no complications and the convalescence was uneventful.

CASE 7 (Fbdy. No. 2638T)—A boy, two years of age, choked while playing with a jackstone. X-ray examination showed the foreign body to be lodged at the suprasternal notch. Within twenty-four hours he was admitted to the Bronchoscopic Clinic. X-ray examination at this time showed the jackstone to be in the same location, but there was marked subcutaneous emphysema in the neck. The child was slightly dyspneic and had considerable difficulty in swallowing. The foreign body was removed without anesthesia with the aid of a child size laryngoscope and cupped forceps by the dangling method. The child was kept under observation for several days until the subcutaneous emphysema subsided. No infection followed the entrance of air into the tissues, and he was discharged well at the end of a week.



(a) (b)

Fig. 2. Case 9. (a) Roentgenogram of chest showing jackstone in the esophagus with a right sided pneumothorax. (b) Same patient, roentgenogram shows empyema of the right lung. Drainage tube in position following thoracotomy.



(a) (b)

Fig. 3. Case 9. (a) Roentgenogram of chest after drainage of empyema at time of esophagoscopic removal of jackstone. (b) Roentgenogram showing the final result after removal of the jackstone. Patient discharged well.

Comment:

1. In this case fortunately infection did not follow the emphysema in the neck.

2. Introduction of a small catheter through the nose and esophagus, allowing it to remain for feeding until the emphysema has subsided, is of value to prevent infection.

CASE 8 (Fbdy. No. 2799T)—A child, five years of age, had her mouth full of jackstones and choked. All of the foreign bodies were regurgitated except one that passed into the upper esophagus back of the cricoid. She was brought immediately to the hospital, where an x-ray examination located the jackstone and showed marked displacement of the larynx and trachea. No attempts at removal had been made. Five hours after the accident the hypopharynx was examined with a laryngoscope and the jackstone was found lodged at the level of the cricopharyngeus. It was removed by the dangling method with cupped forceps. There were no complications and the convalescence was immediate.

CASE 9 (Fbdy. No. 2809T)—A boy, six years of age, choked while playing with jackstones. He was immediately taken to a local hospital, where x-ray examination showed a jackstone in the upper esophagus. Under general anesthesia two unsuccessful attempts at removal were made. After the second examination extensive emphysema of the neck developed and slight dyspnea. At this time the child was referred to the hospital. On admission roentgen examination showed a jackstone in the esophagus at the level of the suprasternal notch. There was present a periesophageal cellulitis and mediastinitis and a right sided partial pneumothorax. (Fig. 3A). The esophagus was examined with the administration of a small amount of thin mixture of bismuth subnitrate and this passed readily alongside the jackstone through the esophagus. There was no evidence of communication between the esophagus and the pneumothorax in the right pleural cavity. The child had considerable fever and because of the pneumothorax he was kept under observation. The pneumothorax gradually increased and a fluid level developed. The child was able to take fluids without difficulty. It was thought inadvisable, however, to attempt esophagoscopic removal of the foreign body because of the danger of producing bilateral pneumothorax from the emphysema already in the mediastinum. The empyema in the right pleural cavity was drained by Dr. Walter Estell Lee through a rib resection. (Fig. 3B.) The child's temperature came to normal, several transfusions having been given during the period of observation.

At the end of four weeks the lung had expanded but there was still some drainage from the fistula in the chest wall. (Fig. 4A.) The foreign body was removed from the esophagus with the esophagoscope. No anesthesia was used. A 7 mm. full lumen esophagoscope was introduced and the jackstone was found embedded in the folds of the upper esophagus. Considerable granulation tissue was present in the location of the foreign body. Fluoroscopic guidance was used so that the behavior of the jackstone could be closely observed when traction was made on an upward projecting globular arm of the foreign body. No undue reaction resulted from the removal of the foreign body. The esophagus returned rapidly to normal and the empyema cleared up gradually. The child was discharged well with a completely healed wound three months after admission (Fig. 4B.)

Comment:

1. The presence of acute unilateral pneumothorax contraindicated further attempt at esophagoscopic removal of the foreign body because of the danger of the pneumothorax extending to the opposite side.

2. Surgical drainage of the empyema made the esophagoscopic removal a much safer procedure.

CASE 10 (Fbdy. No. 2830T)—A girl, six years of age, choked while playing with jackstones. Considerable difficulty in swallowing developed, but no dyspnea. Fifteen hours after the choking attack she was admitted to the hospital, where an x-ray examination showed the jackstone at the level of the suprasternal notch in the esophagus. Without anesthesia a 6 mm. full lumen esophagoscope was introduced and the foreign body was removed with ball forceps by the dangling method. No complications followed the removal of the foreign body.

CASE 11.—An adult choked while having a jackstone in the mouth. X-ray examination shortly after the accident showed it to be in the stomach. No difficulty in swallowing developed. It passed through the bowel without difficulty and was recovered within forty-eight hours.

CONCLUSIONS

1. Jackstones are potentially very dangerous foreign bodies when lodged in the esophagus.

2. Serious trauma in most cases has been due to hasty and ill advised attempts at removal.

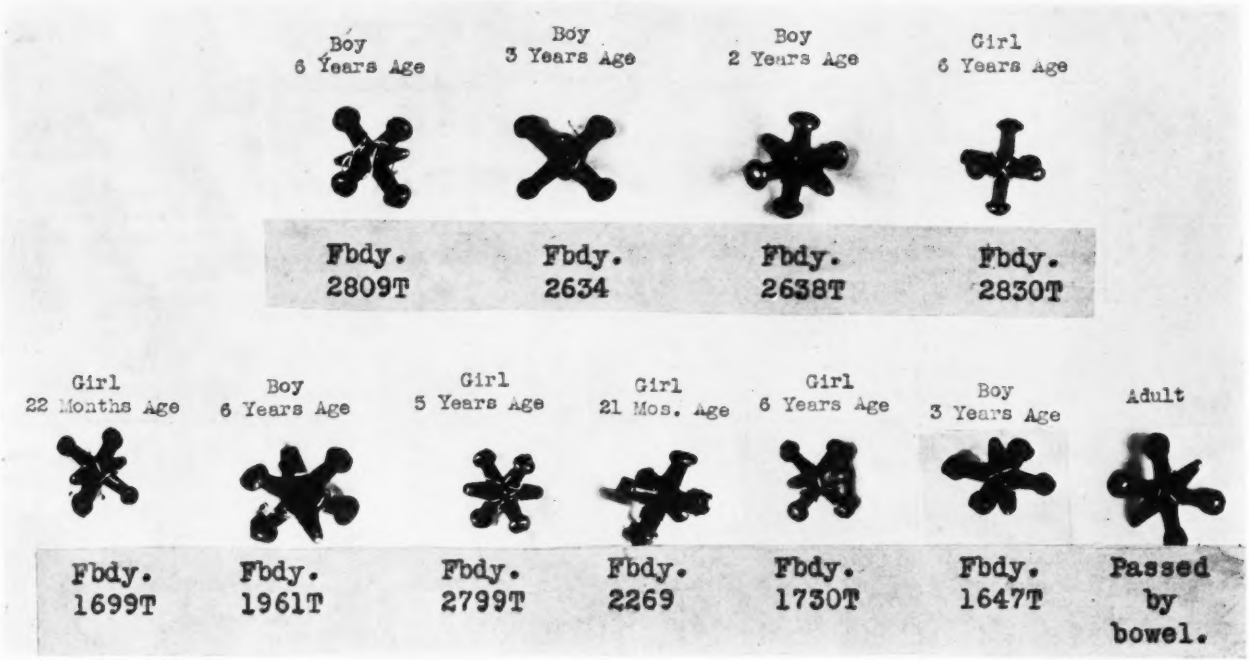


Fig. 4. Photograph of jackstone specimens removed from the upper food passages. Reference to foreign body numbers indicate the jackstone in each case.

3. Urgent dyspnea is the only indication for immediate operative interference, and this should be relieved by tracheotomy. The jackstone can then be removed, after the tracheotomy wound is well sealed off, with safety. Dyspnea may also occur during attempts at removal. This will necessitate temporary discontinuance of efforts at removal and withdrawal of the esophagoscope.

4. The safest method of removal is the dangling method of Jackson. In using this it is most important that the endoscopic tube used be equal in diameter to the greatest diameter of the jackstone. The diameter of the jackstone may be determined by x-ray examination and the matching of a duplicate foreign body. X-ray guidance during removal may be of value in some cases.

5. The presence of unilateral pnemothorax may be a temporary contraindication to the removal of the jackstone from the esophagus. After the pleural lesion is under control the forign body may be removed esophagoscopically without danger of a bilateral pneumothorax.

326 South Nineteenth St.

XXIV

A PIECE OF GOLD BOUGIE (TWENTY YEARS IN SITU) REMOVED FROM THE DISTAL HALF OF THE EUSTACHIAN TUBE DURING A RADICAL MASTOID OPERATION*

HILL HASTINGS, M.D.

LOS ANGELES

A very brief review of the main features in the history of this case is in order.

A man, age 63, consulted me on October 1, 1936, because of purulent discharge from the right ear and severe loss of hearing. He had been deaf in both ears since childhood, due to a bilateral chronic middle ear suppuration which had ceased about twenty-five years ago. He had been treated off and on for this deafness. There had been no other symptoms, except an attack of dizziness, about twenty-four years ago, with a feeling of falling to the right. This disappeared in a day or two.

The present trouble began six or seven weeks ago with a bloody discharge from the right ear and very greatly increased deafness in this, his better ear. It did not follow an acute upper respiratory infection. He was being treated for a chronic sinus disease by another otolaryngologist who had performed bilateral transantral ethmoid operations four years ago with apparent success, as far as headaches and relief of moderate nasal discharge.

The patient brought with him x-rays of the head, made about fifteen years ago, which showed a foreign body, apparently a piece of wire, in the distal half of the eustachian tube. He stated that he persuaded another otolaryngologist, about twenty years ago, to use an electrically stimulated gold bougie to see if the hearing could be improved, and that evidently at the last treatment the piece was broken off in the tube, unknown to him, and likely unknown to the doctor. It is interesting, and quite unusual, that my patient said he

*Specimen and x-ray pictures shown at the Western Section, American Laryngological, Rhinological and Otological Society, January 30, 1937.

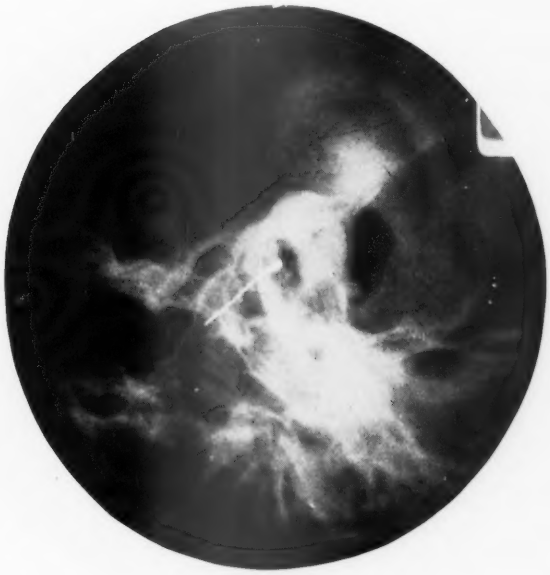


Fig. 1

did not blame the otologist who was responsible for the accident. He stated that he himself had bought the battery and gold bougie and had insisted on its being tried out for a series of treatments.

It is noteworthy that the patient, a very intelligent lawyer, was quite sure that the foreign body had not caused any increased deafness during these twenty years, and stated that this severe deafness only appeared with the bloody purulent discharge six or seven weeks ago.

The examination showed a chronic purulent otitis media in the right ear and a severe loss of hearing to voice and forks. The fundus of the left ear showed a healed condition with thick scarring. Time will not permit a detailed report of my examination notes. No other signs or symptoms were found.

A radical mastoid operation was advised but consent was deferred, as the patient hoped that local treatment would bring back the fairly serviceable hearing that had existed.

After a few days a sudden onset of dizziness occurred on getting out of bed, and he was unable to walk unassisted. I had him

Fig. 2

taken to the hospital. Examination showed a horizontal nystagmus to the left. The loss of hearing was now complete. There was no fever, no headache, no mastoid signs, and no other symptoms. A radical mastoid was then performed. I felt that the labyrinthitis was serous and not yet purulent.

My operative findings were briefly as follows: A sclerosed mastoid; small antrum, containing pus and granulations; the tympanic cavity contained pus and granulations; cholesteatoma in the oval window recess and aditus; caries of a part of the bony wall of the horizontal semicircular canal.

After cleaning out the middle ear cavity and with special care in the region of the orifice of the tube (for fear of pushing the bougie out of reach) the end of the bougie was seen; barely showing at the mouth of the tube. It was removed with ease. The piece is $\frac{5}{8}$ of an inch long.

The recovery has been uneventful, except for facial paralysis, which I believe was due to a severe neuritis from curetting the granu-

lations in and around the oval window recess. The nerve was not exposed to view. Extreme care was taken in the operative procedure, especially so as there was a reasonable fear that the complete loss of hearing indicated a serous labyrinthitis that might be converted into a purulent labyrinthitis.

1136 West Sixth St.

XXV

TRACHEOTOMY TUBE INCORPORATED IN A NECKLACE

WM. F. BREHM, M.D.

FRANKLIN, PA.

A recent development in the field of laryngology is an appliance which is a particularly effective incorporation of a tracheotomy tube in a necklace. This combination is such as to present what appears to be a necklace and which not only carries the breathing tube but renders the incision scar wholly or partially invisible.

The mechanism of the tracheotomy tube is similar to those already manufactured, but the plate has a filigreed cover which fits snugly to the neck and has a universal joint allowing free movement of the neck. It is held in place by a flat white gold necklace which is fit to size and fastens by a catch in the back. The complete thickness of the plate cover is about 3/16 in. compared with the ¾ in. protrusion of the old type of tracheotomy tubes.

The front filigree cover is removable, and is held in position by two ball-and-socket pull catches on either side, facilitating quick removal.

The tracheotomy tube-necklace is made for both inspiration alone and inspiration-expiration. The primary tube and plate are standard for both types. When inspiration alone is used, a shallow cap with flutter valve is placed over the end of the primary tube and the whole is covered by the filigree cover, the air being drawn through the openings in this cover. The secondary tube is omitted in this type but one with a flutter valve can be used if desired.

When both inspiration and expiration are necessary, the same standard primary tube and plate are used, but a secondary tube with a ball-shaped elbow at its outer opening is used. The air passage here is below on the bottom rim of the plate. This is necessary because mucus would soon clog the filigree cover, rendering it unsightly.

The tubes and plate are of nickel, which is nontarnishable and noncorrosive in tissue juices. The necklace is of white gold and the cover is filigree white gold or platinum and set with three diamonds or other stones.

Fig. 1. Tracheotomy tube in a necklace.

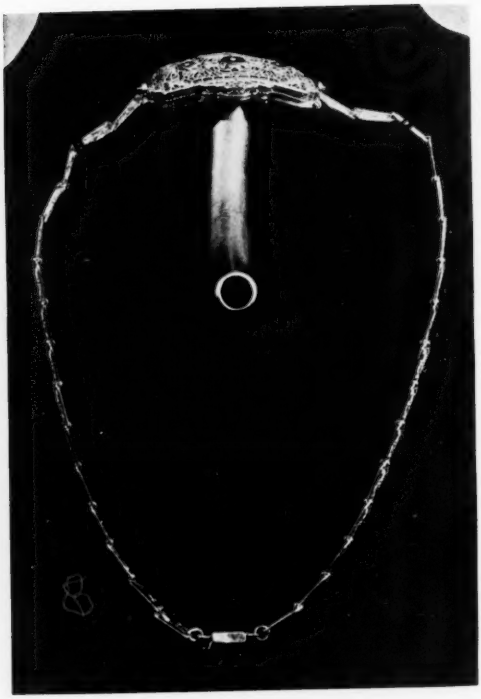


Fig. 2.

The tubes are made in standard sizes and will fit any patient heretofore using the old type of tube. The outside caliber is the same, but the inside is slightly larger, due to the thinness of the wall of the tube. The use of nickel in the manufacture of the tube permits a thinner wall than did the old nickel-plated or silver-plated tubes.

No difficulty has been encountered in the use of the tracheotomy tube-necklace. Its efficiency equals that of the old type of tube and has the added advantage of presenting an object of beauty to the eyes of those who stare at persons unfortunate enough to have to use a tracheotomy tube for a breathing channel.

XXVI

REPORT OF A CASE OF SUBCUTANEOUS EMPHYSEMA CAUSED BY A FOREIGN BODY, A PRUNE SEED IN THE BRONCHUS*

LOUIS DAILY, M.D.

HOUSTON

In the Annals of Otology, Rhinology and Laryngology, of June, 1935, Clerf reports two cases of "Subcutaneous Emphysema as a Complication of a Foreign Body in a Bronchus." Clerf gathered from the literature seven cases which, with the two of his own, made nine cases so far on record of this complication occurring prior to instrumentation. In this series there was only one death—the child died from mediastinal and pericardial suppuration two days following bronchoscopic removal of a peanut. In these cases all the foreign bodies but one were of vegetable origin: a paper clip, a bean, a grain of corn and the rest peanuts. In most of the cases the emphysema extended to the head, neck, chest and mediastinum. In one case it involved the scalp and face, and extended down to the pubis. In the case which ended fatally, at autopsy nothing was found to account for the emphysema. It was thought that a leakage took place along the party wall between the trachea and esophagus. The mechanism of the emphysema is not clear. In the paper clip case the supposition was that the metal perforated the bronchus. Clerf doubts this, pointing out that this complication is rare in metallic foreign bodies in the bronchi such as safety pins, etc. He also maintains that a foreign body of vegetable origin could not injure the bronchus. He believes that the valvular obstructive emphysema causes a rupture of the alveoli along which air escapes into the tissues.

REPORT OF A CASE

My patient, a colored girl, five years old, was admitted to the hospital December 25, 1936. The parents stated that two days previous to admission they noticed a puffiness around the left nipple. This gradually extended over the left side and then over the right side of the chest. At the time of admittance, the subcutaneous emphysema extended over the whole chest anteriorly upwards, over the clavicles,

*Presented before the Harris County Medical Society, February 4, 1937.

to the sides laterally, and down below the region of the diaphragm. It was more marked on the left side. No crepitation could be elicited in the back. Temperature was 101.8; pulse, 130; respirations, 40. An x-ray showed extensive subcutaneous emphysema. The mediastinum, pleura and lungs were normal and negative for a foreign body. The child had coughing spells, and during these spells the emphysema was increased. A tentative diagnosis of spontaneous rupture of the trachea or bronchus was made.

At the request of Dr. Little, pediatrist on the service, I bronchoscoped the child on December 28, 1936. The trachea contained thick mucus. In the right lower lobe bronchus was a smooth, shiny, oval body with its long axis horizontal to the lumen of the bronchus. This tumor-like mass did not completely obstruct the lumen. There was a space in front and behind it. I had great difficulty in removing it, because it was firm, round and smooth and the forceps did not hold it, slipping off. Finally I removed it with a Jackson's medium size ring forceps. I reintroduced the tube and after aspirating a considerable amount of mucus from the bronchus I could make out a longitudinal tear from six to eight mm. in the wall of the bronchus posteriorly and to the left. The tear was blood tinged. I wiped this off a few times, with a cotton sponge, and in a short while the blood reappeared. I applied 10 per cent nitrate solution to the tear, followed by normal saline solution. Temperature at the time of the operation was 101; six hours later it went up to 103. Next day it came down and remained normal. In about six days no crepitation could be made out on the right side of the chest, but there was puffiness and crepitation on the left side. In ten days all the air disappeared from the tissues. The emphysema about the nipples on the left side was the last to disappear. The patient left the hospital in good condition January 9, 1937, twelve days after the bronchoscopic removal of the foreign body.

This case has several interesting features. As far as I know, this is the first case on record of subcutaneous emphysema caused by a prune seed in a bronchus. I have observed a definite break in the wall through which the air most likely passed into the subcutaneous tissues.

While the foreign body was in the right lung, the subcutaneous emphysema appeared first and disappeared last on the left side. I have no explanation for this phenomena.

Usually one removes the foreign body and the tube at the same time, and gives up further manipulation as soon as the foreign body is removed. For the last few years since I have encountered a case

with two foreign bodies in the same bronchus, I formed the habit of reintroducing the tube and re-examining the bronchi and aspirating mucus from the trachea and both main stem bronchi which is usually caused by the irritation of the foreign body. I believe that this procedure often saves the patient pulmonary complications such as bronchopneumonia, edema, etc. In this case it revealed the tear.

1117 Medical Arts Bldg.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, December 7, 1936

DR. IRA FRANK IN THE CHAIR

Mastoiditis in Infants

PROFESSOR HEINRICH FINKELSTEIN, Director Municipal Children's Hospital, Berlin, Germany (by invitation)

(ABSTRACT)

In contrast with the otologists, many pediatricians consider otitis in infancy quite benign; it is supposed to lead to mastoiditis only rarely, and even this, presumably, heals up rapidly after simple incision following formation of a subperiosteal abscess. Finkelstein believes this holds true only for the first six months of life; later, there are not a few serious cases. Of special significance is the occurrence of "latent" mastoiditis without, or almost without, local symptoms; chronic states of fever, septicemia and cerebral symptoms are thereby explained. From a therapeutic point of view, these latent cases are especially important also when pneumonia or dystrophy, lasting for months, are present at the same time. Only suppuration from the ear leads to suspicion of mastoid involvement. Upon operation, antral and peri-antral suppuration are usually found. Drainage results in a tendency to recover from the pneumonia or other conditions ("war on two fronts"). The question of latent otitis as a cause of severe choleriform intestinal disturbances has not been definitely solved, but must be judged with considerable caution. Finkelstein has seen recovery after operation, but in such a small percentage of the cases that the statistics would probably be the same without operation. It may be noted that this subject was discussed in the same way forty years ago.

DISCUSSION

DR. I. A. ABT: It has been a great privilege to come to this meeting and listen to this paper on a very important subject. I wish to say a word about Professor Finkelstein. He has made important contributions on nutrition of infants, he has been a careful clinician,

a logical interpreter, and his writings and teachings have inspired pediatricians in every part of the world. We are fortunate to have received his message tonight.

It would be presumptuous to attempt to discuss before this group any strictly otologic subject, so I will speak of parenteral infection. It has been known for a long time that an infection in any part of the body may affect the state of nutrition of the infant. Otitis, active or latent mastoiditis, pyelitis, furunculosis, or, for that matter, pneumonia, measles, or any other acute infectious process may produce symptoms in the gastro-intestinal tract. Those most frequently observed are vomiting, anorexia, diarrhea, abdominal pain and distention, and loss of weight with dehydration. Everyone who has had anything to do with children knows that anorexia is one of the most striking symptoms of severe digestive disturbance. An infant who does not take fluids will soon show a declining or stationary weight. When this refusal of fluids occurs, whether it is due to an infection of the middle ear or mastoid, of the skin, or to primary intestinal infection, the child is in a serious condition from the outset because his immunity becomes diminished. In addition, these babies have diarrhea. As Professor Finkelstein showed in the chart, they may have thin stools, with consequent loss of water from the bowel. These combined symptoms of anorexia and diarrhea may be caused by parenteral infection, such as active or latent mastoiditis, leading to disturbances of metabolism and abnormal function. Assimilation of food is diminished, water balance is disturbed, and weight disorders occur. Parenteral infection does not mean sepsis; it does not mean that there is a focus from which micro-organisms are being poured into the circulation. It means that there is some form of toxemia arising from local infection.

Goeppert, a student of Heubner and Langstein, and famous for a monograph on otolaryngologic diseases of children, had the idea that when these children vomited as a result of parenteral infection, some of the vomitus was aspirated into the eustachian tube and consequently the child developed otitis or mastoiditis. Professor Finkelstein said that forty years ago otologic diseases of children were already being discussed. Ponfick, a student of Recklinghausen and assistant to Virchow, showed a long time ago that children who had died of nutritional disease or acute infections, such as scarlet fever or measles, at autopsy showed otitis media and sometimes mastoiditis, whether this had been recognized during life or not. In this country Marriott revived the subject of parenteral infections. He was much interested in parenteral origin of diarrheal diseases. He had the idea

that in infantile atrophy after severe diarrhea babies became secondarily infected from vomiting, much as Goeppert had taught, and he said that, in autopsies of infants dying from mastoid infections, colon and Morgan dysentery bacilli were recovered. The Morgan bacillus belongs to the typhoid-dysentery group found in severe diarrhea. Those cases which Goeppert and Marriott would make secondary to gastro-intestinal disease are probably secondary to toxemia caused by a focus of infection, producing gastro-intestinal symptoms and fever. It may be assumed that restlessness, crying and loss of sleep use up a great deal of bodily energy and predispose to loss of weight and lowered resistance. These babies lose water, not only because of severe diarrhea, but also through vomiting and exhalation from the lungs. In consequence, the immunity is being constantly diminished. It has also been shown that in babies suffering from parenteral infection and consequent gastro-intestinal symptoms the hydrochloric acid of the gastric content is diminished, and food is retained in the stomach a long time. Fever may result from the focal infection, parenteral infection, or from an infectious process in the intestinal tract. Professor Finkelstein showed long ago that there may be an alimentary fever quite independent of intestinal infection. He has also shown that in these children forced feeding does not correct the stationary or declining weight.

The cause of parenteral infections is not easily explained, but every clinician knows that in a certain proportion of middle ear infections, mastoiditis, or in other focal infections, anorexia, loss of weight, fever and diarrhea do occur.

The treatment was fully covered by Professor Finkelstein. If the source of infection can be recognized it should be treated surgically. The difficulty lies in recognizing the source of infection, though if the focus can be located radical procedure is indicated and it should be carried out promptly. It has been thought that breast-fed children are less liable to parenteral infections than artificially fed infants, though this is not always true. Babies in homes are perhaps less disposed to these parenteral infections than those in institutions. It may be largely a question of immunity. A properly balanced diet should be given, including the necessary vitamins. We do not know which vitamins are needed to raise immunity, though their administration tends to maintain a normal nutritional state.

DR. FRANCIS L. LEDERER: A world renowned teacher does us the honor of presenting a phase of pediatric otology which, some years ago, was subject to great controversy in this country. Numerous attempts have been made to link otogenic sepsis and alimentary intox-

ication or atypical dysentery into a disease entity so as to form a new clinical, therapeutic and epidemiologic foundation for the serious nutritional disturbances occurring in small children. The literature still contains sporadic reports of a particular type of otitis media and mastoiditis occurring in malnourished infants, but now the plea is mainly with the pediatrician who states "It is sometimes difficult to convince the otologist that there is a mastoid infection and get him to carry out the rather simple procedure indicated." (C. H. Hall, J. Oklahoma State M. A., 29:247 (July), 1936.)

While in the main Professor Finkelstein's presentation deals with this type of case, which he chooses to term "latent" because no classical sign of surgical mastoiditis is present, he has likewise presented other clinical types of mastoiditis. For example, the 12-year-old boy who was not operated upon because there was no pain or swelling behind the ear. This is a frequent experience because certain men are unwilling to admit surgical mastoiditis until the auricle assumes an angle of 90 degrees to the side of the head. Such cases, however, cannot be judged latent and surely would evidence other signs of surgical mastoiditis, such as sagging of the posterior superior canal wall, poor hearing, roentgenologic evidence, temperature, discharge, in addition to a history of a process extending over a period of several weeks. In this connection we recognize atypical varieties which must be considered, such as the mucosus capsulatus course of otitis media and mastoiditis, the pneumococcus or phlegmonous type and those which are accompanied by evidence of intracranial extension, especially to the venous sinuses and meninges. We must, therefore, also remember individual, seasonal bacteriologic, anatomic and yearly variations in ear disease. The terms "latent," "silent" or "masked" have appeared in the literature a number of times in describing mastoiditis. Some years ago H. L. Williams (Minn. Med., 15:813 (Dec.), 1932) described such a form in which the appearance of other more general symptoms took precedence over the ear symptoms which in themselves were not too classical.

To many of us, otitis media of infants has been brought home primarily as an anatomic problem. With this fundamental knowledge in mind, we have been able to evaluate and adequately treat inflammations of the middle ear of infants. A statement to the effect that complications of otitis in the first year of life are not as frequent as in older children is quite true, but certainly they are more common than generally admitted. I need not dwell at this time on the difficulties of otoscopic interpretations of the tympanic membranes of infants because of the narrow lumen of the canal and the

horizontal position of the drum. An important factor in the accessibility of the middle ear of infants for infection is the size and angulation of the eustachian tube, but even more important in the pathogenesis of middle ear inflammation is the presence of mesenchymal tissue. Mesenchyme absorbs slowly and sometimes not completely, making possible isolated areas of infection. Such abscesses may take many weeks to coalesce. Furthermore, we must understand that the tympanic membrane of infants is thicker and more elastic than that of the adult and may therefore withstand greater intratympanic pressure. It is for this reason that Dr. Finkelstein finds subperiosteal abscess so frequent in infants under three years of age, the petrosquamosal suture being less resistant than the tympanic membrane.

Professor Finkelstein, despite his many, many years as a teacher and clinician, is perturbed by the seeming lack of accord and variance of opinion concerning clinical mastoiditis. In my comparatively short otologic experience, I have ceased trying to reconcile the experience of the local pediatrician who in thirty-five years of practice never encountered a surgical mastoid, with that of my own. If I do get into a controversial discussion over indications for a procedure, whether it be paracentesis or antrotomy, I want to know first if we are both considering the same fundamental pathologic entity.

There are two possible reasons for finding thirty-two occult cases out of a total of ninety-five cases of infant mastoiditis. The first is for the anatomic reasons which have already been mentioned, and this possibly could be obviated by closer study of the ear and the elimination of all other foci. The causes of failure in diagnosis have been set forth by A. A. Love (Arch. Otolaryn., 17:297 (March), 1933) as (1) insufficient history; (2) failure to associate distant manifestations with otitis; (3) lack of proper evaluation of minor anatomic changes in the external canal and tympanic membrane, and (4) insufficient follow-up of potentially dangerous cases. The second reason is prompted by the vagueness of signs and symptoms which all of the exponents of antrotomy for nutritional disturbances of infants have willingly admitted. By surgical intervention in febrile, nutritional cases without ear symptoms or findings, Professor Finkelstein finds his indications justified merely on the basis of recovery. It is said that these cases do not develop classical signs because of the lack of resistance to develop local reaction. In the light of our knowledge this hardly seems plausible. Where there is ample anatomic, pathologic and clinical evidence to substantiate the claim that there is infection within the mastoid, the profound systemic effect in infants is unquestionable. Those cases with discharging ears and sepsis which

were operated upon no doubt were justified as well as those with meningitic manifestations, but it is most difficult to enthuse over indications for operation in cases of cholera morbus which were operated upon without ear findings. Such infants who succumb have secretion in the tympanum. Thereby rests the contention that the focus was located here, despite the fact that the majority of infants' middle ears may contain an exudate on postmortem examination, even though they have not died of general nutritional disturbances.

We can readily understand the relationship between analogous lymphoid structures of the pharynx and the intestinal tract, but it would be most generous to credit the middle ear with similar possibilities as a potential source of infection.

The same questions come into my mind as were aroused in the essayist's as to the difference in material seen, whether it be in social class or geographical distribution, but at the same time where clinical entities call forth such a liberality of interpretation, they are possessed of grave dangers and pitfalls. This has been amply demonstrated by the experience of a few years ago. For the interpretation and evaluation of latent ear infections I can but recommend more careful histories and examinations on the part of pediatricians and otologists.

DR. ALFRED LEWY: Following up Marriott's work on the relation of nutritional disorders and latent mastoiditis, and following conversations with Dr. Wishart of Toronto, who read a paper on this subject before the American Otological Society, and Dr. Lyman of St. Louis, my associate, Dr. Hagens and I, as attending otolaryngologists at the Children's Department of the Cook County Hospital, undertook, beginning some five years ago, to arrive at some conclusion. We had the utmost co-operation from the pediatricians at the hospital and with Professor Jaffé, the pathologist. There are 250 beds, including fifty to seventy-five for infants, and while we did not personally examine all the ears of diarrhea cases, the residents did. They are well trained in otoscopic examination, and any case that appeared suspicious was held for us to examine.

We did not undertake to operate upon any case that did not show some otologic indication, not even paracentesis. This does not necessarily mean a red drum membrane, for secretion may exist behind a pale one, and for mastoid indications we would expect a discharging ear, swelling of the posterior canal wall or other evidence.

We have come to the conclusion that there is no benefit in doing antrotomy without an otologic indication. The postmortem examina-

tions have shown no overlooked cases, and the small amount of secretion occasionally found in the tympanic cavity, according to the pathologists, could have no influence on the outcome. Bacteriologic investigation of this secretion at necropsy is impracticable.

There were, during this period, two epidemics of diarrhea among the newborn, one of them attributed by the bacteriologic department to B. pyocyaneus and the other to streptococcus hemolyticus. In none of these that came to necropsy were ear complications discovered.

Dr. Hagens has a number of temporal bones not yet completely examined, and I would like to have him give his point of view.

In the five years or so there have been two deaths from otogenic complications that developed after entrance to the hospital. All the other fatal cases had their fatal complications upon entrance and were so diagnosed, which speaks well for our carefulness. On the other hand, we have cleared up persistent pneumonias by doing antrotomies which we believed indicated. The residents are instructed to look with particular care for evidence of otitis media in pneumonias, and, in fact, all illness not otherwise accounted for requires otologic investigation.

Dr. E. W. Hagens: I thing we have tried to follow out what Professor Finkelstein has said. In the cases accompanied by high fever, where no cause is found, we are open minded. If the ear is discharging and infected we are willing to try to obtain better drainage, even if the mastoid infection is not as definite as we would wish. We recently opened an antrum under local anesthesia and the child made such a nice recovery that the pediatricians think there is something to it. We provided, perhaps, better drainage to the middle ear and antrum in that way. We also agree to operate in sepsis where there are no other findings, and we agree to operate where there are intracranial complications when no other source is found. We are willing to provide as good drainage as possible in those cases. In focal infection of the ear and lung and kidney we are willing to help out if we think we can do some good. In most cases of pneumonia it has been felt that this disease was causing most of the trouble and we could not help by operating on the ear.

In regard to the ears we are working on out at the University, they are of a little different type. I tried to get some temporal bones of the children I had seen before death in whom there was no reason to open the ear or the antrum. Unfortunately, I did not get those

temporal bones and do not know what they contain. I was interested in the cases where we found no indications for operation and the child died, and I wished to see if we had missed an infection. The pediatricians do not find many of that type. They look at them and satisfy themselves that there is nothing in the ear, and at autopsy they do not find sufficient evidence in the ear, nor does Dr. Jaffé find sufficient pathology to tell us to be on our guard. We have had very little material to go on that would support the idea that the ear is the cause of the trouble.

Histologic work-up of temporal bones from children dying of otitis media with intracranial complications will often show bilateral inflammation when the history was more or less unilateral. I think the history will usually show that there has been an otitis on the apparently unaffected side, but this has cleared up before we see the patient. Yet histologic examination will show that there is infection there and that the pneumatic spaces are all infected. I do not know whether that means that we should have operated. I think many cases go along with infection of the pneumatic spaces, and of the marrow in some instances, that we do not know about. As to infection from the petrous bone, I think that will have to be worked out as time goes on. When a child dies from meningitis there was probably an ear involvement that may have gone through the petrosa, but it is not easy to find the infection there. It is true that the petrosa is infected, but the leads to the meninges are not so common.

Dr. Louis W. Sauer: It is a great privilege to be allowed to come here and listen to your distinguished guest. I can contribute very little to the excellent paper Professor Finkelstein presented, unless it might be a word about our experience at the Cradle in Evanston, where, for six years, infants came in, apparently well, only to die within two or more weeks after admission. Soon after admission, signs of enteritis developed and at autopsy mastoiditis was found. Year after year such cases occurred. When Professor Finkelstein was in Chicago some years ago he visited the Cradle. He was shown these charts, which were, in many instances, very similar. Charts of one year would be practically identical with charts of the following year. At that time his explanation for these cases, after examining the charts, was "infection." We could not decide then which came first, whether the enteritis or the mastoiditis. Conditions became worse, in spite of customary precautions. In 1927 the authorities were tempted to close the Cradle because of this high incidence of infection. Cases operated upon for mastoiditis had the same results as cases not operated upon.

In 1927 there were eighty-seven cases with twenty-seven deaths. The autopsies were performed by Dr. J. L. Williams, at the Evanston Hospital. He, with Dr. Gladys and Dr. George Dick, made very careful pathologic and bacteriologic studies. Dr. Williams has told me that in this series he felt there was no question that the enteritis came first. We became convinced that he was right; in retrospect we are sure he is right. They isolated the Morgan dysentery bacillus not only from the intestinal canal, but also from the mesenteric lymph glands, which were somewhat enlarged. Upon injection into rabbits the cultures produced the same symptoms; even the filtrate of the cultures produced symptoms of enteritis.

An aseptic nursery technic has been in effect at the Cradle since this severe epidemic. Dr. Gladys Dick, a director of the Cradle for many years, has solved the problem of cross-infection in infant nurseries. She proved that the spread of enteritis came through contamination and that mastoiditis was secondary, or occurred in the late stages of the disease. The Morgan bacillus was found in the mastoids; sometimes the colon bacillus was also found in the mastoids. After the proper bacteriologic technic was evolved, the Morgan bacillus was found constantly.

The technic is different from any used elsewhere for infants. Although infants come in, occasionally, with enteritis, cross-infection enteritis has not occurred. No infant has been lost from cross-infection mastoiditis, otitis media or enteritis since the technic was evolved. Should you encounter a case of this type in any nursery, it is well worth while to insist on this absolutely prophylactic technic, which has worked at the Cradle 100 per cent for more than eight years.

DR. T. C. GALLOWAY: No laboratory experiment could have been devised which would seem to settle the question of mastoiditis with epidemic enteritis more definitely than this particular one which the Cradle was having. Cultures for the Morgan dysentery bacillus were positive from the blood, from the stools, and from the mastoid in most instances. In no case was this organism found in cultures of the nose and throat. These cases had all the classical symptoms Marriott pointed out as the mastoid syndrome. Especially did they have the diarrhea and a falling weight curve, and I think the scientifically controlled work in this epidemic by the Dicks would seem to show quite positively that the mastoiditis was entirely incidental, was not a primary cause and had no bearing on the outcome of the case. I operated on five of them and it appeared to make no difference at all

in the clinical course. A trained otologist in these cases should recognize that the ear drum is not quite normal.

An antrotomy in infants a few months old is very simple, can be done under local anesthesia without pain or shock, and if indications are present antrotomy should not be refused. Especially should this be done in the other type of cases described by Professor Finkelstein.

Dr. Joseph C. Beck: I wish to express by appreciation in hearing Professor Finkelstein speak tonight. I do not think, from my own experience, that I can add to the subject, and if it were not for the letter I hold in my hand from Professor LeMeé of the Hôpital Enfants Malades, Paris, I would not accept our President's invitation to enter into the discussion. I think only the otologists who have a service in connection with pediatrics can give us any information on this subject from the otologic standpoint. In this letter Professor LeMeé sent me last week he makes the statement that in his experience with this type of disease, having followed the literature very carefully throughout the various countries and especially in France, in the majority of cases antrotomy was done, as stated by Dr. Galloway. That it was so simple that one could easily err in saying that the correct indication was perhaps not present, but the results proved that it was the proper thing to do, namely, perform antrotomy. He speaks particularly of the embryonal tissue found in the middle ear and mastoids in infants, which is so subject to infection and from which so much absorption takes place. I think some of you will remember Professer Schmiegelow, of Copenhagen, when he visited this country a few years ago. During the evening we had an informal discussion and asked him about this new disease we were discussing at that time, namely, diarrhea as a symptom of mastoiditis in infants. Professor Schmiegelow laughed and said they had discarded those operations long ago, that it made no difference, as was stated by Dr. Galloway, in the majority of the cases. I think also Dean's work at Iowa City and later in St. Louis with Professor Marriott should be mentioned here. They very definitely put themselves on record regarding the otoscopic picture, and operated upon only those cases where they were certain there was likely to be an infection in the ear. I do not know how these enthusiasts feel now, but when you ask otologists now about these cases they say "the pediatricians make us do a good deal we do not like to do."

Dr. Samuel Salinger: Since the pediatrician in recent years has been armed with the otoscope, we otologists have had less opportunity to examine the ears of infants and young children than for-

merly. Nevertheless, those of us who are connected with children's hospitals or general hospitals with a pediatric service are occasionally called in to see the type of case under discussion. The request is usually put this way, "Will you please examine and tell us whether a mastoid operation is indicated?" Being trained in the otologic school we frequently find the usual indications of a mastoiditis to be absent and therefore say "no." However, on the insistence of a pediatrician that there must be a focal infection in the mastoid we reluctantly yield and perform the operation. I have seen this type of case in which there were no surgical indications for mastoid operation, as we have been taught them, which nevertheless at operation revealed mastoid pathology and were followed by subsidence of the gastro-intestinal symptoms. On the other hand, I have operated and found minimal pathology and achieved no results so far as the principal ailment was concerned. The difficulty from our point of view is that we see these patients after the acute otologic symptoms have subsided and are not in position to judge what the true otologic picture may have been. In many of these cases it is necessary to observe the drum membrane throughout the course of the infection in order to be able to draw conclusions as to its type and severity.

My standpoint on this matter is that, if the history is clear, the symptoms following a definite respiratory infection, I am willing to assume that there is mastoid infection, even though the usual local signs are lacking. The cases are not in the same class as the epidemic type of gastro-intestinal upset described by Dr. Sauer. I claim that we do see patients who previously were in good condition, with a good appetite and normal state of nutrition who suddenly begin to develop loss of appetite, loss of weight, diarrhea, vomiting and so on, which are due entirely to a focal infection in the mastoid or the middle ear, and it is this type of case that will recover if operated upon promptly.

Professor Heinrich Finkelstein (closing): I believe that a new and most important point of the subject is the surprisingly favorable effect of operation for latent mastoiditis upon recovery from chronic pneumonia and pyuria. The diagnosis of otitis in severe diarrhea is made difficult by the absence of swelling and hyperemia, as a consequence of persistent dehydration.

Abstracts of Current Articles

NOSE

Communications Between Subarachnoid Spaces and Nasal Mucosa (Sulle comunicazioni fra spazi sub-aracnoidei e mucosa nasale).

Novelli, D. (Firenze), Boll. delle Malatt. Orecch. Gola, Naso, 51:131 (April), 1936.

Experiments carried out along the same line by numerous investigators are herein discussed.

In some instances india ink or prussian blue was injected into the cisterna magna and the lumbar regions, while in others radiopaque fluid was inserted into the subarachnoid space. In the latter the quantity of the fluid used was controlled by x-ray.

Those who have carried on these investigations are of the opinion that colored substances injected into the subarachnoid space first pass into the perineural areas and later into the lymphatics of the nasal mucosa.

In this respect Novelli has found that injections of colored substances into the cisterna magna too often produce distressing symptoms both during and after the procedure, and consequently the animals frequently died. Secondly, because of the remote position of the atlanto-occipital membrane from the area under consideration, an excess of fluid must be employed with the result that the inevitable rise in pressure causes alterations in the surrounding tissue. Hence, by the use of a short-pointed needle, the author injects a few drops of Ringer's solution, containing 5 per cent india ink, into the meninges covering the olfactory bulb. Since the above mentioned region is accessible in young rabbits with a minimum of distress, the author prefers to carry out his experiments upon them rather than upon any other animal. Moreover, it may be added that the method of injection employed has produced no ill effects on the rabbits, either during or after the injection. The animals are destroyed one week later.

At the gross examination the injected area had acquired a slate color, while the same area on the opposite side was not discolored. The nasal mucosa corresponding to the operated side had a deep slate color, particularly on the superior portion at the olfactory region, while the nasal mucosa on the opposite side was normal.

The microscopic examination of the superior turbinate disclosed the granules of india ink to be completely enclosed in the perineural membrane and hence, strictly localized in the perineural space. The accompanying photomicrograph demonstrates this clearly.

These examinations proved that the independent lymphatic network and the remaining portion of the nasal mucosa had no pigmentation whatever. Further, it reveals that the colored substance had been transported by the current of arachnoid fluid in the perineural spaces found in the olfactory mucosa and was not forced by pressure, since the pigments would, in that case, be present in the nasal mucosa.

SCIARRETTA.

Presence of a Histamine-Like Substance in Nasal Mucosa, Nasal Polypi and Nasal Secretion.

Buhrmester, Catherine C., and Wenner, W. F. (St. Louis), Arch. Otolaryng., 24: 570 (Nov.), 1936.

Nasal polypi and normal mucous membrane are shown to contain a histamine-like substance in approximately equal amounts in the moist tissue. Calculated on the basis of the dry weight of the tissue, polypoid tissue contains more histamine than the normal membrane.

Nasal secretion from persons with allergic rhinitis does not have depressor activity when injected intravenously. The hydrolyzed secretion contains much less histamine-like substance than do polypi, calculated on equal protein values.

Histaminase is not present in allergic nasal secretions.

It is suggested that the increase in mineral content of the nasal discharge, with its resulting effect on permeability, difference in potential and acid and acid-base equilibrium, are factors to be considered in studying the formation of polypi and other allergic manifestations.

TOBEY.

The Biological Method of Surgical Cure of Ozena.

Doroschenka, I. T. (Dnieperostroi), Acta Oto-lar., 24:2:315, 1936.

After separating the mucoperichondrium of the nasal septum to form a canal, an appropriately shaped piece from the last costal cartilage with its perichondrium on one side is secured under local anesthesia and inserted; no sutures, vaseline gauze pack. Excellent results after three years; no absorption and no failures of the graft to remain in place.

FENTON.

Surgical Treatment of Frontal Sinusitis and Its Complications (Considerações sobre o tratamento cirurgico da sinusite frontal e suas complicações).

de Sanson, R. D. (Rio de Janeiro), Rev. Otolar. de S. Paulo, 4:279 (Jul.-Aug.), 1936.

Using the external radical operation of Jansen-Ritter and commonly attacking the ethmoid as well, de Sanson agrees with Furstenberg that radical and extensive bone removal is imperative when osteomyelitis supervenes. Frontal lobe abscess is entered under a large osteoperiosteal flap by diathermy puncture, and this puncture is enlarged weekly, if necessary for a month or more, until the capsule is sufficiently solid to permit complete exsection of the abscess. The greatest care must be taken to avoid rupture during removal. Twelve cures are reported.

FENTON.

PHARYNX.

Agranulocytosis and Amidopyrine.

Dyke, S. C. (Wolverhampton), Brit. Med. J., Nov. 7, 1936, p. 911.

Attention was first drawn to agranulocytic angina by Schultz in 1922. He outlined a new clinical syndrome consisting of the association of angina, in the sense of ulceration of the throat, with a diminution or disappearance from the blood of the white cells of the granular order. Since that time cases have been recorded in increasingly large numbers. Agranulocytic angina occurs much more commonly in Germany, Denmark, and America than in Great Britain. It shows a peculiar incidence as regards medical men and those associated with the medical profession.

The etiology of the disease is discussed. The ingestion of amidopyrine and of its close allies containing a benzene and a substituted pyrazolone ring is capable, in sensitive subjects, of giving rise to agranulocytosis. It is evident that sensitivity to the drug is confined to certain individuals whom, in the light of present knowledge, we have no means of identifying beforehand. The writer points out that the newer analgesics and hypnotics have had greater popularity in the countries mainly affected. The subjects are almost entirely past the prime of life and it is suggested that sensitivity to the drug is in some way associated with a change in the nature or balance of the sex hormones.

The mechanism of production of agranulocytosis remains obscure. The rapid diminution of the white cells in the circulatory blood after the administration of a test dose of amidopyrine to a sensitive individual is striking; a marked fall within from two to three hours has

been found. The obvious explanation of this rapid disappearance is a destruction in the blood; examination of the blood, however, fails to reveal any damaged cells or other evidence of their destruction. Beck has suggested an arrest in the process of maturation of the cells in the bone marrow, but it is difficult to see how a mere arrest of maturation could bring about so sudden a fall in the circulating leucocytes. Pentnucleotide was introduced as a means of stimulating maturation of the leucocytes, and in normal subjects it seems to be effective in this respect. However, the evidence goes to show that in agranulocytosis following on the use of amidopyrine it is ineffective.

The writer reports three cases of agranulocytic angina, in all of which amidopyrine or an allied drug had been administered before the onset of symptoms.

GOLDSMITH-MCLEOD.

Some Tumors and Ulcers of the Palate and Fauces: Sir Felix Semon Lecture.

Howarth, Walter (London), The Lancet, Nov. 14, 1936, p. 1139.

Palatal tumors are not so uncommon as is often supposed, and it is surprising what a large variety occur. The writer points out that this region lends support to Cohnheim's theory, that tumors originate from embryonic rests, as it is one in which relics of fetal tissue may be expected, for the palate is formed by a complicated folding in of fetal structures.

Mixed tumors, which are by far the most common ones that occur in the substance of the palate, are discussed. The method of origin of these tumors has given rise to controversy, but most authors believe that they are epithelial in origin and arise from fully developed glandular tissue. These tumors are for the most part very slow growing and it is not uncommon for them to be present for many years before discovery. They are usually encapsulated and are generally regarded as comparatively benign in character. This, however, is not always the case and, particularly if the capsule is ruptured through trauma or if an incomplete operation is performed, the growth invades the tissue surrounding it, undergoes malignant changes, and may give rise to metastasis.

Hemangioma and hemangiofibroma are rare tumors in the palate, but it is advisable to consider the possibility of their occurrence, as in two cases that the writer knew of an incision was made, with almost fatal results, in the belief that a peritonsillar abscess was present. The writer has seen only one fatty tumor in the palate and the

number of cases that have been recorded is very small. There was one case of adenoma and one of adenocarcinoma in his series of cases. Osteomata have been occasionally described in the palate. The writer had one case with a hard oval bony swelling under the mucosa of the hard palate in the midline. It proved to be an example of "torus palatinus," an anatomic peculiarity consisting essentially of a projection downwards of the diploe covered over by compact bone.

In malignant disease of the palate there is usually an ulcerating tumor or raised plaque with a varying amount of infiltration around. But in one type of epithelioma there is a diffuse shallow ulceration with a serpiginous outline and little or no infiltration at the edge. This resembles tuberculous ulceration; it is not very painful and the malignant nature is often not suspected until a piece has been removed for microscopic examination. The superficial invasion of a wide area and the fact that different portions show varied characteristics is typical.

Apart from ulceration associated with cancer, syphilis and tuberculosis, some streptococcal infections may be very chronic and resistant to treatment, more particularly those that are due to streptococcus viridans. The writer believes one form of shallow ulceration is a definite entity which he has described under the designation "precancerous epitheliomatosis"; fifteen or twenty years may elapse before definite malignant changes supervene. Repeated microscopic examination in atypical and slowly progressive ulcers might result in more definite diagnosis and the application of a more efficient form of treatment.

In operations inside the mouth the writer prefers the diathermic knife to the scalpel. The absence of bleeding gives a remarkably clear field. In malignant tumors the depth of penetration of the spark insures the destruction of a wider area of tissue than that removed, and the risk of distributing cancer cells in the wound is less than with scalpel and forceps. In the writer's series of 61 cases of malignant disease of the palate and fauces, 25 per cent were alive more than five years after operation. Of the remaining cases, 36 per cent died within the first year, while the remaining 39 per cent lived for periods varying from one year to four years. These figures may not seem very satisfactory but many of the cases were regarded as hopelessly inoperable in the ordinary way and were complicated by malignant glands in the neck which necessitated block dissection of one or both sides.

GOLDSMITH-MCLEOD.

Malignant Tumors of the Cricopharyngeal "Belt" (Tumeurs malignes de la "ceinture" crico-pharyngée).

Ducning, J. and L. (Toulouse).

Rejecting all surgery of the retrocricoid and periesophageal region when growths are recognized, because of inoperable extension into deep pharyngeal lymphatics, these authors state that the radium collar or the Coutard fractionation of deep roentgen therapy are the only available treatments, but that the prognosis is always grave—usually six months or less after recognition. Most of these growths are spinocellular with pearl formation. While tumors of the pyriform sinus near by are almost exclusively masculine, one-third of these cricopharyngeal growths are found in women.

FENTON.

ESOPHAGUS, TRACHEA AND BRONCHI

Indications and Technic of Endobronchial Application of Radium (Indications et technique des applications endobronchiques de radium).

Mounier-Kuhn, P. (Lyons), Bronchoscopie, Oesophagoscopie et Gastroscopie, No. 4, Oct., 1936, p. 347.

The author reports the use of radium in five patients for the treatment of endotracheal and endobronchial tumors and for tracheobronchial hemorrhage due to mucosal bleeding without specific disease. His technic consists in using 25 mg. of radium bromide in gold filtered tubes. These are placed in the region of the lesions after thorough anesthetization and previous bronchoscopic orientation. For the trachea, a soft esophageal sound is used, the radium fixed to its end and the entire apparatus left in place. For the bronchi, the tube is introduced under direct vision, and is tied to a long double thread by which its position is maintained and withdrawal made easy. The patient is kept absolutely quiet and under morphin. Each treatment lasted two hours and they were repeated every other day. No untoward reactions occurred and the results were promising, especially in the hemorrhage cases. The author feels that even with this limited experience the method is worthy of trial.

KLAWANS.

Importance of Bronchoscopy in Pulmonary Tuberculosis (Importance de la bronchoscopie dans la tuberculose pulmonaire).

Myerson, Mervin C. (New York), Bronchoscopie, Esophagoscopie et Gastroscopie, No. 4, Oct., 1936, p. 352.

In the past, bronchoscopy has rarely been done in proved or definitely suspected cases of pulmonary tuberculosis. This paper is based upon a series of 150 such examinations, done in carefully selected

chronic cases of pulmonary tuberculosis where the symptoms, physical signs and x-ray findings left some doubt as to their interpretations. Acute involvement in the active stage and hemoptysis within seven to ten days are regarded as contra-indications to the procedure.

The indications in tuberculosis are the same as in the nontuberculous, viz., obstructive lesions, hemoptysis of unknown origin, asthmatic and asthmatiform breathing, localization of lesions for the information of the thoracic surgeon, persistence of bacilli in the sputum despite adequate surgery, and to obtain cultures and smears from secondary bronchi.

Lesions in the tracheobronchial tree are similar to those in the larynx and may be proliferative or destructive with secondary cicatricial obstruction in the latter. Peribronchial lesions may also be recognized from endobronchial changes. The author describes the various lesions found in this series and appends illustrative case reports.

In only two cases did hemoptysis follow bronchoscopy, and these were cases with frequent hemorrhages before the examination. No bacillus-negative case has become positive as a result of bronchoscopy nor has a quiescent case ever been activated. The author feels that bronchoscopy is a valuable and safe procedure in properly selected cases of pulmonary tuberculosis.

KLAWANS.

EAR

Acute Streptococcal Infections of the Middle Ear.

Mollison, W. M. (London), Brit. Med. J., Sept. 26, 1936, p. 615.

In this article the writer really includes all acute infections of the ear, because the streptococcus is the infecting organism in the vast majority of cases. Although it is possible for the ear to be involved in the course of a septicemia of nasal as of any other origin, ordinarily the inflammation reaches the ear, either by continuity along the mucous membrane to and along the eustachian tube or by discharges from a septic focus in the nose being blown through the eustachian tube into the tympanum, without involvement of the intermediate tract of mucous membrane. By far the commonest cause of acute ear infection is an acute infection of the whole upper respiratory tract; should the antrum become infected, ear infection is more likely.

Dr. Lowndes Yates has pointed out that mucus was an antiseptic and that if the mucus was removed or diluted it failed to keep the

organisms in check. Two important ways by which the nasopharyngeal mucus is so diluted is by bathing in fresh-water baths and by using nasal douches.

Lowered resistance due to other factors doubtless paves the way to streptococcal infection of the ears, and it is suggested that the virulence of the streptococcus may be enhanced by the presence of another micro-organism, or by the virus of the common cold or influenza. Persons who are found to carry hemolytic streptococci in the throat or nasopharynx are less susceptible to streptococcal infections. A Dick test will show whether a patient is susceptible to the hemolytic streptococcus, and these patients can be immunized against this organism.

The severity of the illness of patients with acute streptococcal infections varies enormously. Regarding operation on the mastoid the writer believes that in many acute toxemic cases it is far better to tackle the patient instead of the ear, the procedure being to take cultures, to give the appropriate serum and even to group the blood with a view to possible blood transfusion. Blood tansfusions and pentnucleotide injections mark a great advance in the treatment of severely ill cases of infection.

GOLDSMITH-MCLEOD.

The Problem of Otosclerosis.

Chubb, Gilbert (London), Brit. Med. J., Dec. 12, 1936, p. 1192.

The clinical picture of otosclerosis is so indefinite, its symptoms are so often shared by other forms of deafness, and its physical signs are so few and inconstant that the diagnosis can only be made with varying degrees of probability. The writer points out that the clinical picture of otosclerosis need not possess one symptom that is not constantly present in other and far more common types of deafness. The outstanding pathologic features of otosclerosis are: the bony fixation of the stapes, the occasional involvement of the basal coil of the cochlea, and, though this has not yet been proved, degenerative changes in the auditory nerve. According to the relative time incidence of these factors, the clinical picture of otosclerosis may apparently vary from that of a middle ear "obstructive" deafness to that of pure nerve deafness. At the best, the Bezold triad can only be of diagnostic value in uncomplicated cases in which fixation of the stapes precedes any serious involvement of nerve or cochlea. Yet it is made to apply to cases in which otosclerosis is supposed to co-exist, as well it may, with conditions known themselves to produce the

Bezold triad—that is to say, middle ear deafness or loss of low note perception.

Certain other features of otosclerosis are discussed, and it is noted that they may all be absent in otosclerosis, while with very few exceptions, even their presence only becomes significant in the aggregate. Therefore it is not surprising to find that the proportion of deaf patients diagnosed as suffering from this disease differs greatly with different observers. The personal equation of the observer bulks largely in the uncertain diagnosis.

Various treatments are discussed but there is no treatment known to affect the course of the disease. The writer emphasizes the need for a scientific approach to the problem of otosclerosis, which is not likely to be solved by doubtful diagnosis and still more doubtful observations on the results of treatment. Gray's treatment by injections of thyroxin has been very disappointing.

GOLDSMITH-MCLEOD.

Dural Nerves of the Posterior Cranial Fossa and Their Importance in Diagnosis and Surgery of Petrositis (I nervi della dura madre della fossa cranica posteriore e la loro importanze nella diagnosi e nella chirurgia delle petrositi).

Porta, C. F. (Parma), Arch. Ital. di Otol., Rino. and Laring., 48:389 (May), 1936.

The article contains a discussion of the symptomology of petrositis as presented by Kopetzky and Almour, Eagleton, Ramadier, Brunner, Ruskin and many others. Also a detailed analysis of various anatomic investigations describing the nerve distribution in the dura of the middle and posterior fossæ. Of the latter, he emphasizes mostly the work of Arnold, Burger, Dowgjallo and Fenton.

The author conducts his researches on 12 cadavers (3 children and 9 adults). None of these individuals had ever suffered from intercranial diseases and the specimens were removed shortly after exitus. Each specimen consisted of the dura mater, which covered the entire petrosa, the cerebellar and cerebral fossæ, the tentorium and falx cerebri. Details of the technic for preparing this material are given.

The results of his researches proved that the nerves supplying the dura of the posterior cranial fossa derive from two sources: the trigeminal and the vagus.

The trigeminal innervates the dura with branches derived from its ophthalmic division. These branches are divided into two groups.

The first consists of numerous minute filaments originating almost where the ophthalmic nerve branches off the Gasserian ganglion and supply the dura covering the anterior surface and apex of the petrous bone. The second group is composed of one or two nerves, also called the recurrent nerve of Arnold. Porta finds the distribution of these to be very irregular, therefore he separates them in four distinct types and describes them in detail.

The vagus supplies the dura only with the posterior meningeal nerve. The extent of its distribution is of two varieties.

Such a great difference in the topographic distribution and morphologic diversity of these nerves changes the symptomology of petrositis. These facts explain the reason for the presence of all the diagnostic symptoms, such as retrobulbar, ocular and occipital pain, in some patients, while in others only one of these pathognomonic signs is evident and yet the petrosal involvement is identical.

Finally, the author concludes by stating:

1. That the retrobulbar pain should be attributed to the small fine branches of the ophthalmic nerve.

2. That the occipital pain is due to the nerve of Arnold and the meningeal branch of the vagus.

SCIARRETTA.

Some Uncommon Forms of Vertigo of Vestibular Origin (Sopra alcune forme non comuni di vertigine d'origine vestibolare).

Battistelli, G. F. (Trieste), Arch. Ital. di Otol., Rino. and Laring., 48:331 (May), 1936.

Under the term uncommon, the author eliminates all the classical lesions as well as all destructive involvement of the labyrinth; Ménière's disease, trauma and hemorrhages of the labyrinth including syphilitic and rheumatic lesions of the eighth nerve; but he refers to those forms of vertigo in which the location of the lesion and etiology are uncertain and hence difficult of diagnosis.

In these cases the otoscopic examination and the vestibular reactions are normal, but the patients are seized by a severe vertigo, vomiting and sometimes fainting upon sudden movement of the head or upon assuming certain positions. When these patients are examined during attacks a nystagmus is observed. The duration of this symptomatology usually extends over a few weeks when it gradually disappears either with or without therapy.

The author considers this illness to be caused by a lesion in the vestibular centers of the medulla arising from an exogenous or endogenous intoxication following a ptomaine poisoning, some infectious disease or allergic condition.

The therapy is complete rest in bed, diuresis and diaphoresis and, when indicated, desensitization.

The article is completed by the presentation of three interesting cases.

SCIARRETTA.

Contributions on Middle Ear Suppuration and Its Complications Resulting From Enterococcus Infection (Nuovo contributo allo seudio delle suppurazioni timpaniche e complicanze da enterococco).

Fornari, G. B. (Cantania), Arch. Ital. di Otol., Rinol. and Laring., 48:355 (June), 1936.

The literature is reviewed with due credit to Citelli as being the first to call attention to the destructive nature and sometimes fatal termination of an enterococcic infection of the middle ear and the brilliant results obtained by vaccinotherapy.

The author stresses the importance of establishing an early etiologic diagnosis and the early use of vaccine as the means of saving many lives. The difficulty of the bacteriologic research is that it requires an enormous amount of laboratory work.

Fornari has made extensive investigations both of the clinical and the laboratory phases of this subject and presents a short cut to the clinician and technician.

A search for the enterococcus should be made under the following circumstances:

1. When the patient presents an acute or an exacerbation of a purulent otitis media in which the bacteriologic examination of the secretion discloses an extremely varied morphology of the invading bacteria. (Isolated cocci, in pairs, unequal size, arranged in chains, etc.)

2. When the morphology of the enterococcus is so similar to the pneumococcus that differentiation is impossible. The prognosis of these patients is grave.

3. When patients (not affected by constitutional diseases) gradually become worse even with the best of care.

The writer recommends that cultures be made from the aural discharge of these patients on an ordinary agar medium. After twenty-four or forty-eight hours subcultures are made on the following media: first, peptone water; second, 20 per cent bile; third, esculin; fourth, Hiss' with mannite. The remainder of the growth is used for the manufacture of autogenous vaccine.

Six cases are reported with the following conclusions:

The symptomatology of an enterococcal otitis media is similar to other types yet it does not respond to the best approved medical treatment.

Grave complications develop easily in these types of infection and usually are not controlled by the best surgical procedures.

The early use of autogenous vaccine is successful because the enterococcal infection diminishes the power of the body defenses against the invading organism.

SCIARRETTA.

Hemorrhages Following Paracentesis of Tympanic Membrane; Frequency and Significance of Jugular Fossa Dehiscence in the Tympanic Cavity (Le emorragie da paracentesi della membrana timpanica e la frequenza ed il significato della deiscenza della fossa guigulare nel cavum tympani).

Federici, F. (Genova), Arch. Ital. di Otol., Rino. and Laring., 48:145-183 (March), 1936.

The author reports a severe hemorrhage following accidental injury of the jugular bulb after paracentesis of the left tympanic membrane. The patient recovered without complications, and bleeding was controlled with a firm gauze packing through the external auditory canal.

Hemorrhages following paracentesis of the tympanic membrane are the sequences of surgical injuries of the internal jugular vein or carotid artery, projecting abnormally in the tympanic cavity through dehiscences in its ventro-lateral wall and absence of the posterior wall of the carotid canal.

This uncomfortable experience prompted Federici to investigate the frequency of such an accident. These hemorrhages are relatively rare because in the literature reviewed there were only eighteen cases due to injury of the jugular bulb and one of the internal carotid. Recovery took place in all cases that were treated by packing. Two died of pyemia following an electrocautery paracentesis. In the third, a diabetic subject, the bulb was injured while performing a mastoidectomy; pyemia developed and terminated fatally.

Judging from the number of paracentesis performed, hemorrhage should occur more often. However, since the number of cases recorded is so small, the author is of the opinion that, in the majority of instances, there is an uneventful recovery with the consequence that the average otologist does not consider the condition sufficiently serious or important to be worth reporting.

At the Anatomical Museum of the University of Genova he diligently examined 1,228 skulls, that is, a total of 2,456 temporal bones (79 were of children less than 6 years of age). He found 36 dehiscences in 34 skulls (bilaterally in two skulls). Some of the openings were small and only 10 heads had dehiscence in the jugular fossa of such a dimension that (in life) the vein could project far enough in the tympanum to be easily injured. Hence, the existence of such anomalies of jugular fossa are 4 per 1,000 and of the carotid canal 0.4 per 1,000. He also observed dehiscences six times on the medial wall of the fallopian canal (about 2.6 per 1,000).

Federici finds the percentage of the anomalies in his series much lower than that reported by other writers. This becomes more evident when one considers that the total number of skulls examined by him is approximately the same as that reported by all other investigators.

Finally, the author seeks a phylogenetic explanation for the presence of dehiscences in this location. He discusses theories advanced by previous writers, presents an extensive critical study of the comparative anatomy and embryology of the structures under discussion and concludes by offering a logical theory.

SCIARRETTA.

Pneumatocele of the Mastoid Region Following Mastoidectomy (Pneumatocele della regione mastoidea in soggetto mastoidectomizzato).

Ferraris, G. (Torino), Arch. Ital. di Otol., Rinol. and Laring., 48:251 (April), 1936.

The oddity prompts the author to report this case.

A patient who had been operated upon ten months previously for a right, acute, simple mastoidectomy with complete closure of the wall presented himself to the doctor and complained of intense pain with the following symptoms:

After the operation the patient had been well up to fifteen days prior to his appearance, at which time he had contracted a severe head cold. Tumefaction in the mastoid area, accompanied by excruciating

pain in the ear and right side of the head, had developed within twenty-four hours following a forceful blowing of the nose.

Examination revealed that a large diffuse tumefaction had developed in the entire retroauricular region that extended throughout the mastoid area and pushed the auricle forward. This tumefaction was fluctuating and painful on palpation, though there was no erythema of the skin. The tympanic membrane was inflamed and bulging and the hearing was impaired; temperature was normal. The patient was sent home to return for operation at the hospital the following day.

Instead of the expected flow of fluid upon incising, a blow of air was expelled. Inspection of the cavity revealed that it was deep, perfectly smooth and dry, pearl gray in color and covered with normal thin epithelial membrane. The wound was closed by suture.

On the following day the patient was in excellent condition; hearing was normal, the tympanic membrane also normal, with no evidence of bulging. The patient was dismissed and cautioned to be careful in blowing the nose.

Evidently the author considers the possibility of the formation of a circular scar at the aditus or at the antrum which acts as a valve in one direction. Such could easily occur in this case because, when the mastoidectomy was performed. the antral region was found to be full of granulation. As customary in instances of such pathology, he curretted this area thoroughly.

SCIARRETTA.

Cancer Metastasis in the Temporal Bone Simulating Acute Inflammation in the Middle Ear.

Nylén, C. O. (Copenhagen), Acta Oto-lar., 24:2:242, 1936.

Three years after breast amputation, a woman of 64 developed acute otitis in one ear, with but little discharge on paracentesis and some radiographic blurring. Continued pain and swelling above the ear led to a mastoid operation, finding normal cells except for "granulations" from the antrum through the tegmen and root of the zygoma. This proved to be carcinomatous. Death followed from multiple metastases after temporary amelioration of hearing and of swelling and pain about the ear.

Operation being only of temporary aid and metastases usually multiple, it is suggested that radium or deep irradiation therapy might have been preferable in such a case.

FENTON.

Contributions of Andreas Högyes (Über die litterarische Tätigkeit des weiland Andreas Högyes).

Rejtö, A. (Budapest), Acta Oto-lar., 24:1:103, 1936.

The work of Professor Högyes (1847-1906) included telephonic determination of hearing (1878) and research on nystagmus and vestibular function (1879-1885), culminating in demonstrations of the bilateral ocular movements conditioned by sounding tuning forks, rediscovered by Preyer in 1900 and Agazotti in 1904. Antedating Bárány and others by a generation, Högyes' work remained untranslated from the Hungarian until after his death. In the opinion of Tweedie, Högyes should be joined with Flourens and Ewald as the trinity upon whose work all labyrinthology is based.

FENTON.

MISCELLANEOUS

Pathogenic Fungi.

Whalen, Edward J. (Hartford, Conn.), Arch. Otolaryng., 24:436 (Oct.), 1936.

Fungous infections of the ear, nose and throat and respiratory tract are being observed and reported in increasing numbers.

A routine is suggested for the simple, rapid examination of material harboring the more common fungi.

The similarity between the pulmonary lesions produced by the tubercle bacillus and by some of the pathogenic fungi is noted. The possibility is suggested that the tubercle bacillus may be a yeastlike organism in some phase of its life cycle.

A survey is made of the literature describing the fungous infections of the ear, nose, throat and respiratory tract.

TOBEY.

The Staphylococcus in Relation to Sinusitis, Bronchitis and Bronchiectasis.

Woodward, Fletcher D. (University, Va.), Arch. Otolaryng., 24:753 (Dec.), 1936.

The frequency of infection with the staphylococcus in sinusitis and early bronchiectasis is noted, and treatment along specific lines with staphylococcus toxoid and antitoxin is advocated, in addition to the older, well recognized methods. A brief review of the bacteriology of the staphylococcus is given.

Future work along this line, both in the research laboratory and in clinical practice, is urged.

TOBEY.

Treatment of Hemorrhage in Nonhemophilic Patients With an Estrogenic Substance.

Heinberg, C. J. (Pensacola), Arch. Otolaryng., 24:758 (Dec.), 1936.

The treatment of hemophilia with ovarian extract was demonstrated by Birch to be of value.

Treatment with estrogenic substance is of value for certain types of nonhemophilic hemorrhage following surgical procedures when there is apparent imperviousness or failure of the platelets to break down and liberate thromboplastin, which unites in turn with antithrombin and allows clotting.

TOBEY.

Nasal Secretion and the Thyroid (Sécretion nasale et glands thyroide).

Chavanne, L. (Lyons), O. R. L. Internat., 20:653 (Oct.), 1936.

A long series of experiments on dogs demonstrate energetic limitation of nasal secretion when thyroxin is injected intravenously. Thyroidectomy seemed to increase the amount of nasal secretion, which could readily be reduced by systematic injections of thyroxin.

FENTON.

Simple Achlorhydric Anemia, Plummer-Vinson Syndrome, and Carcinoma of the Mouth, Pharynx, and Oesophagus in Women.

Ahlbom, Hugo E. (Stockholm), Brit. Med. J., Aug. 15, 1936, p. 331.

In this article, the close relationship between simple achlorhydric anemia and Plummer-Vinson's syndrome or dysphagia associated with anemia is discussed. Some writers have expressed the opinion that they constitute two somewhat different forms of the same disease. There are clinical features common to both forms. The patients are almost always females, usually between 15 and 50 years of age, and often they give a history of weakness and anemia of many years' duration. They usually state that the teeth were lost early in life and that fissures or "eczema" appeared periodically at the corners of the mouth. The lips are thin and the opening of the mouth is small and inelastic. The changes in the lips are undoubtedly of the same nature as the atrophy of the mucous membrane which one usually finds in the mouth and pharynx in these patients. The atrophy is most clearly seen on the tongue, which in typical cases is completely smooth. The dysphagia is in many cases so slight that it requires a very careful questioning in order to establish its presence. Patients who have had a rather marked degree of difficulty in swallowing for twenty or thirty years before admission have regarded their inability to take more than "small swallows" more as an individual peculiarity than as a disease symptom.

An analysis of 150 cases of women with squamous celled carcinoma of the mouth, pharynx and esophagus at Radiumhemmet, the radio-therapeutic clinic of the Stockholm Cancer Society, is given. In about 70 per cent of the cases there was signs and symptoms of simple achlorhydric anemia or Plummer-Vinson's syndrome. The distinct relationship which appears to exist between these syndromes and carcinoma probably has its foundation in the atrophic changes in the mucous membrane which are associated with the anemic conditions. The writer emphasizes the great importance of diagnosing these two anemic conditions, not only to treat the disease as such, but to attempt to forestall the development of carcinoma, as they constitute excellent examples of conditions which are precancerous.

GOLDSMITH-MCLEOD.

Notices

The Medical School of Washington University has announced two courses in Otolaryngology for graduates.

The first is an eight months' course for those beginning the study of diseases of the ear, nose and throat. The course begins October 11, 1937.

The second is an intensive four weeks' course for otolaryngologists. The course begins April 12, 1937.

Address the Registrar, Washington University School of Medicine, Scott and Euclid avenues, St. Louis, Mo.

The second annual post-graduate course offered under the combined auspices of the University of Oregon Medical School and the Oregon Academy of Ophthalmology and Oto-Laryngology will be held in Portland the week of April 5, 1937. Dr. C. S. O'Brien of the University of Iowa will conduct the work in Ophthalmology and Dr. W. J. McNally of McGill University of Montreal that in Oto-Laryngology.

Further information can be obtained from the chairman of the Committee on Arrangements, Dr. A. B. Dykman, Medical Dental Building, Portland, Oregon.

A postgraduate course in operative surgery on the cadaver has been announced by the Departments of Otolaryngology and Anatomy in the Medical College of the Univeristy of Cincinnati. Dr. Max Halle, of New York, will be a guest instructor. The dates for the course are May 24 to June 4, inclusive. This is just prior to the meeting of the Examining Board and the American Medical Association.

The George Washington University School of Medicine, Washington, D. C., will give an intensive course in Ophthalmology, May 31st to June 5th, inclusive.

Beside a series of lectures offered by a visiting staff and the local department, there will be an afternoon at the Army Medical Center and Ophthalmic Division of the American Registry of the Army Medical Museum, and an afternoon at the National Bureau of Standards where optical problems will be studied.

Address Dr. Wm. T. Davis, 927 Seventeenth St. N. W., Washington, D. C.

Professor Georges Portmann will conduct an intensive course in Oto-Rhino-Laryngology at the University of Bordeaux, France, beginning July 26, 1937. The course will be given in the English language. Inquiries should be addressed to Dr. James A. Flynn, 1511 Rhode Island Avenue N. W., Washington, D. C.

Books Received

Intralaryngeal Epitheliomas (Les Epithéliomas Intra-Laryngés.)

Jean Leroux-Robert. Paper. Octavo, 172 pages with 61 figures. G. Doin, Paris, 1936.

This excellent monograph is based on sixty cases operated upon by Dr. Hautant, either by laryngectomy, hemilaryngectomy or laryngofissure, and studied clinically, radiographically and as to pathologic anatomy. Serial sections establish the starting point and mode of invasion of each growth, thus explaining the various radiographs and succession of symptoms. Subglottic growths or those hidden under the epiglottis or buried under the mucosa, are often mapped out as to extent by radiographs and the appropriate operative measure is thus more accurately selected.

FENTON.

Nursing in Diseases of the Eye, Ear, Nose and Throat.

By Members of the Staff of the Manhattan Eye, Ear and Throat Hospital, New York City. 12mo of 288 pages with 93 illustrations. Sixth Edition. Philadelphia and London: W. B. Saunders Company, 1937. Cloth, $2.25 net.

This little book is a practical manual for nurses, in the eye, ear, nose and throat operating rooms and at the bedside. The type is clear, the style concise and the photographic illustrations are excellent.

The nurse, however, must be reminded that the operating room and table arrangements are routine at the hospital mentioned, and that elsewhere she will encounter many variations in practice as well as in instruments. Obviously these cannot be described in a small handbook.

The section dealing with the preparation of surgical dressings leaves something to be desired in the way of completeness, but otherwise the book seems adequate.